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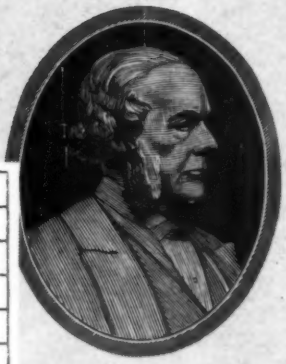

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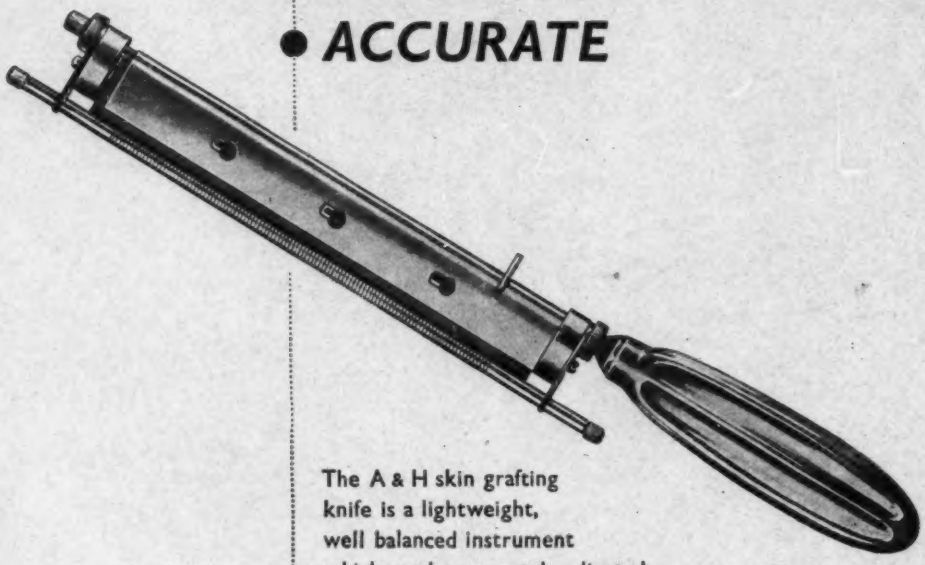

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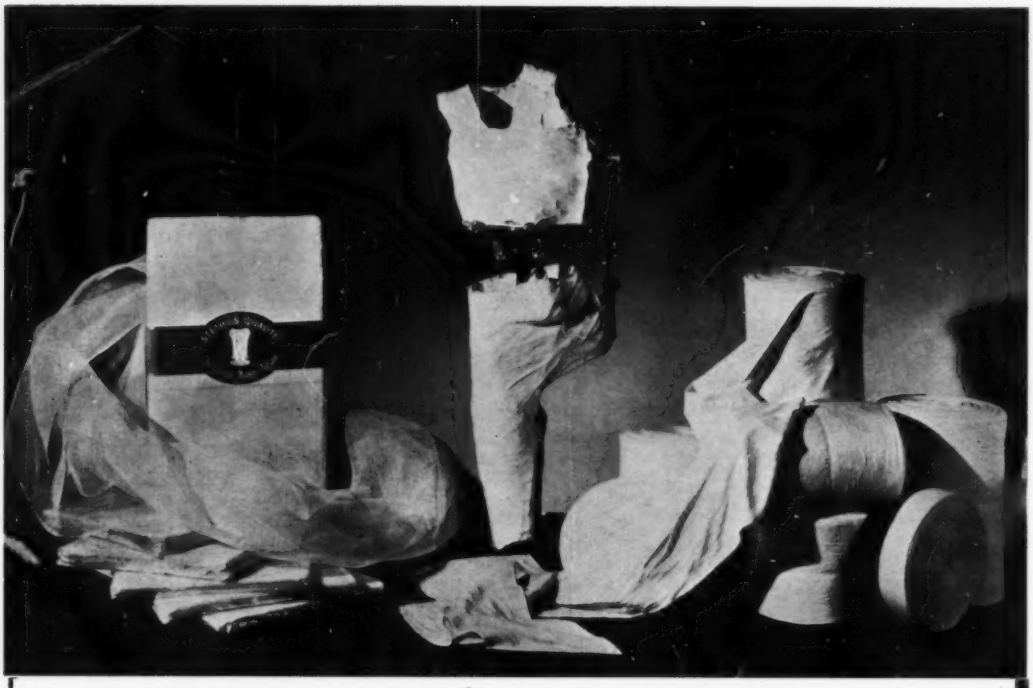

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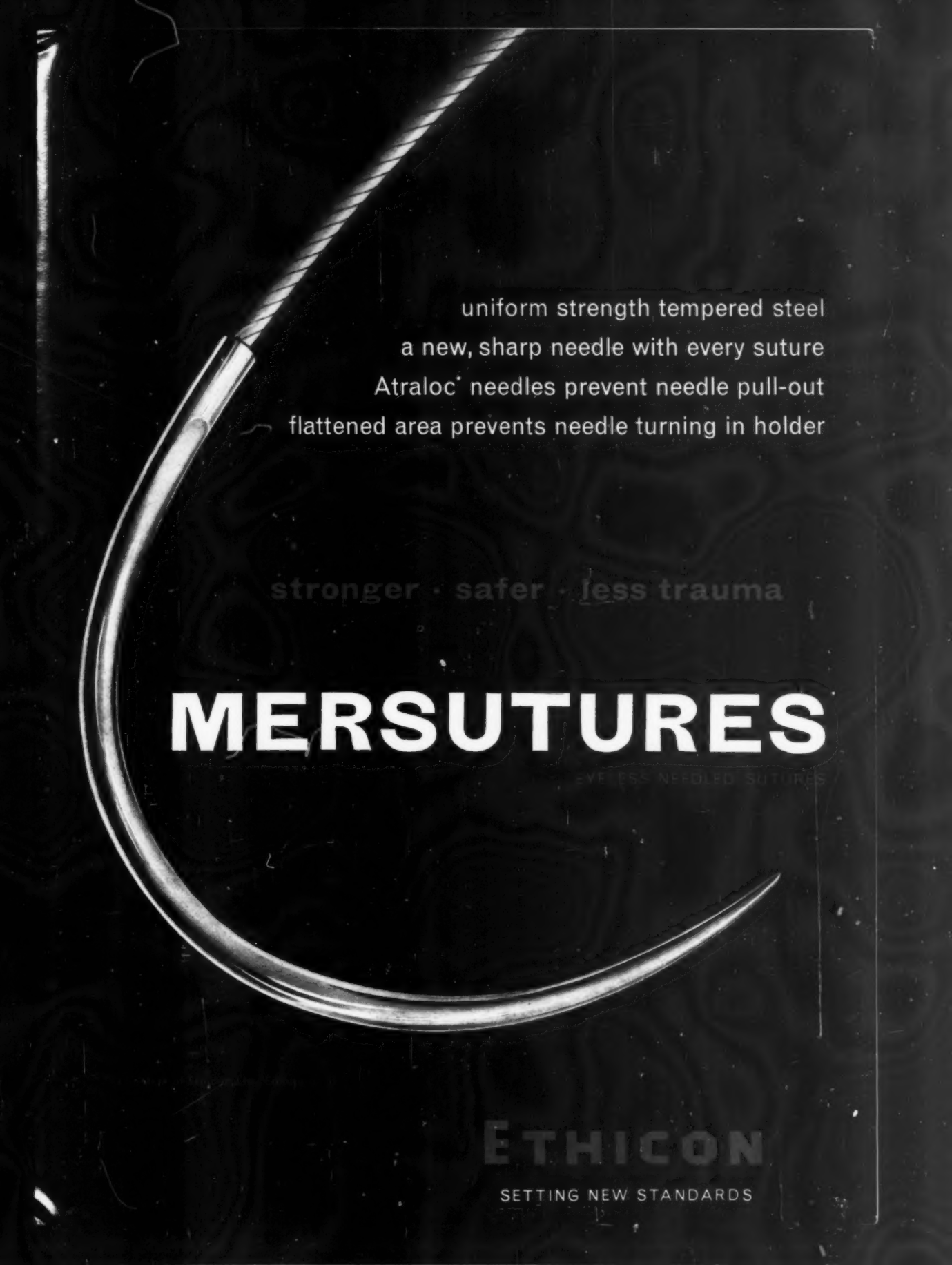
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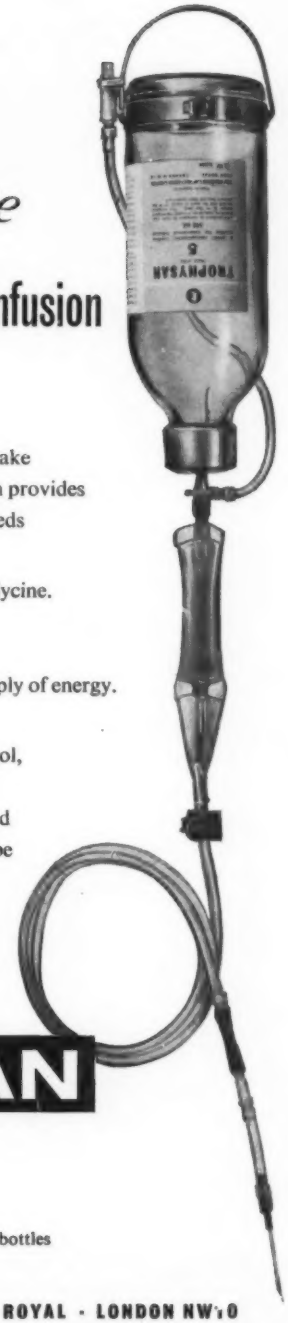

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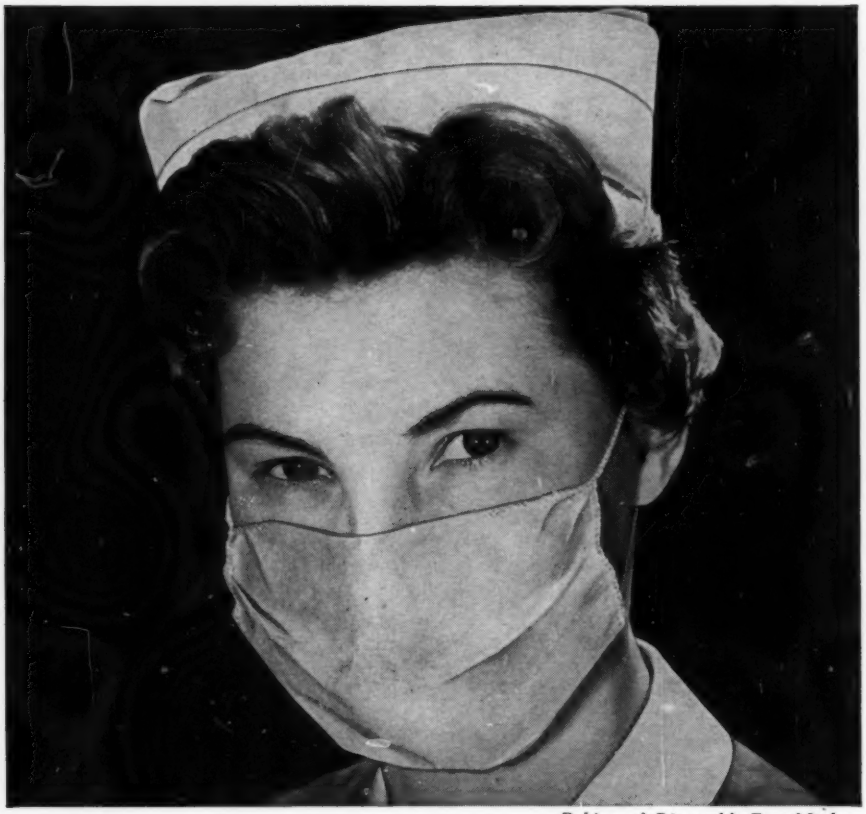

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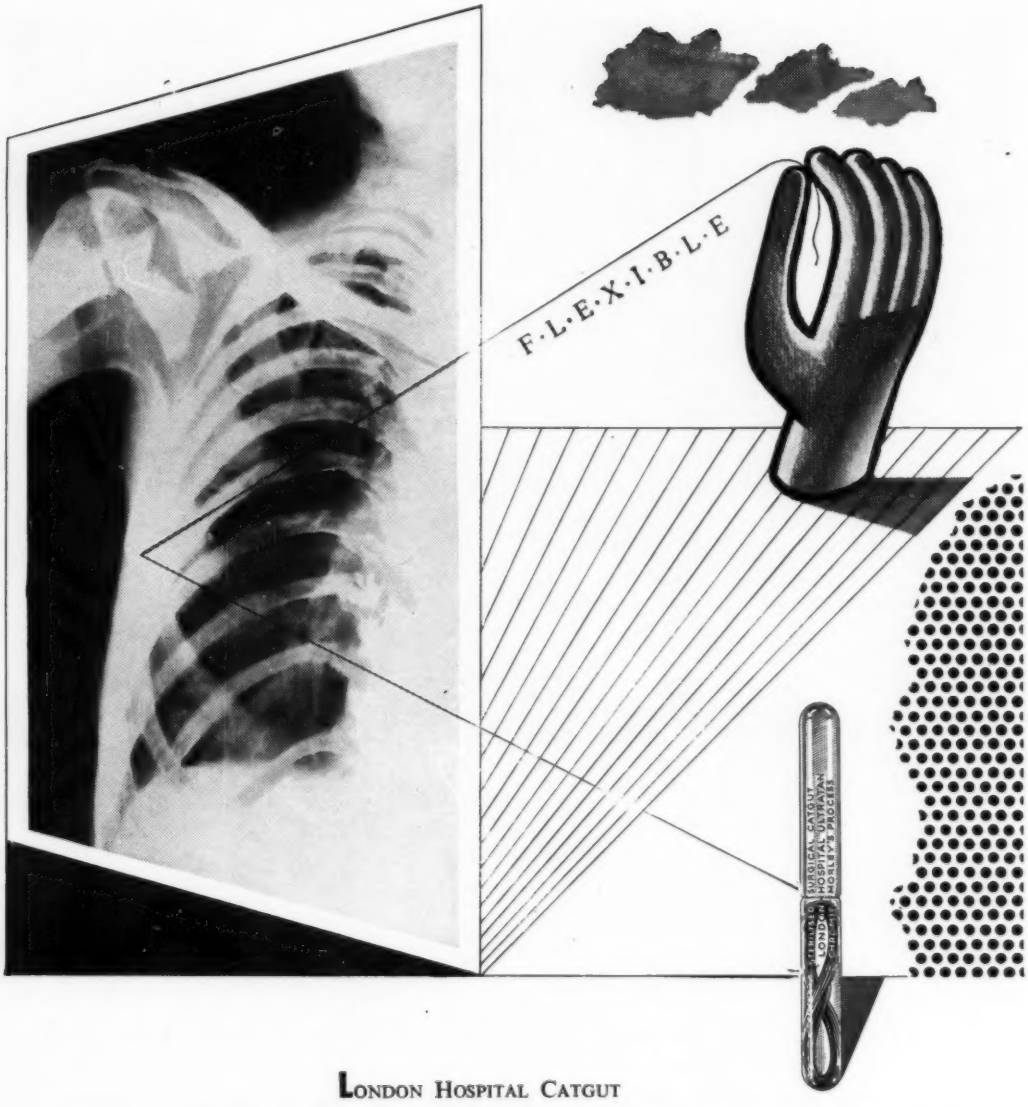

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HERNIATION THROUGH CONGENITAL DIAPHRAGMATIC DEFECTS

BY J. A. W. BINGHAM, BELFAST

SINCE Sir Thomas Dunhill's Arris and Gale lecture on diaphragmatic hernia was published in 1935 little has appeared in British literature on the subject of herniation through congenital defects of the diaphragm, apart from reports of single cases. Views on the types of diaphragmatic hernias that can be classed as due to congenital defects have been clarified a good deal since then, and the results of surgical treatment have improved considerably. This is particularly true for infants with congenital hernias through posterolateral defects of the diaphragm, for in this condition the mortality-rate, even with surgery, used to be distressingly high.

It may be of value, therefore, to review a personal series of 14 cases of herniation through congenital diaphragmatic defects and to discuss the management of such cases. Developmental aspects will not be touched on in this paper, for any discussion of these, to be of value, would need to be of considerable length. The so-called eventrations of the diaphragm will not be discussed, nor will herniation through the œsophageal hiatus. Though hernias of this type are sometimes congenital, they do not, unless in extremely rare instances, result from part of the diaphragm being missing.

TYPES OF DIAPHRAGMATIC DEFECTS

The congenital defects of the diaphragm that I have seen in association with diaphragmatic hernias have been of three types: (1) Posterolateral defects. (2) Retrosternal defects. (3) Extensive defects involving most of the hemidiaphragm. While a few other hernial defects may occur, they do so with exceptional rarity, and it would appear to be generally agreed to-day that these three types constitute virtually all congenital hernial diaphragmatic defects.

Posterolateral Defects.—These defects are also known as 'foramina of Bochdalek', as a result of the paper by Bochdalek in 1848 on congenital diaphragmatic hernias in which he considered mainly congenital hernias in "the posterior portion of the costal part of the diaphragm". Defects in this posterolateral part of the diaphragm are situated with notable constancy at the periphery of the diaphragm in the region of its attachment to the tenth and eleventh ribs. As shown in *Fig.* 1, the outer part of the defect may lie at the point of attachment of the diaphragm to the eleventh rib, while in other instances it lies between the diaphragmatic attachments to these two ribs. The defects occur much more commonly on the left side, and are usually quite small, perhaps 2 or 3 cm. in diameter in infants. In the great majority of posterolateral defects there is free communication between the peritoneal and pleural cavities. Less often, there is a thin hernial sac. This varies a great deal in size in different cases and consists in large part only of pleura and peritoneum.

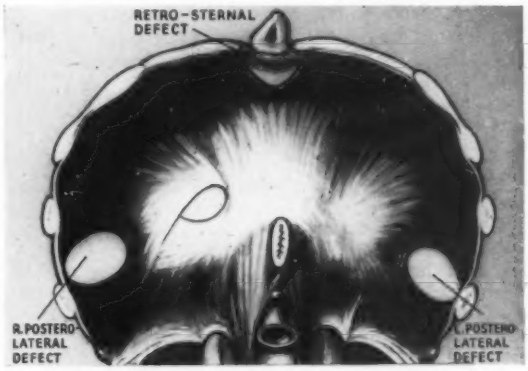


FIG. 1.—Drawing showing the sites of congenital posterolateral and retrosternal diaphragmatic defects.

However, in the two examples of this condition that I have operated on in which hernial sacs were present, the muscle of the diaphragm did not come to an abrupt end at the commencement of the sac, but gradually thinned out and continued for a short distance into the wall of the sac between the pleural and peritoneal layers.

Retrosternal Defects.—The term 'foramen of Morgagni' is often applied to any defect in the anterior or retrosternal part of the diaphragm. Not every hernial aperture in this part of the diaphragm is, however, exactly the same, either in situation or in method of origin. There seems to be no doubt that some hernias in the retrosternal region are acquired, herniation occurring—perhaps late in life—through relatively weak areas, such as between the costal and sternal attachments of the diaphragm (Boyd and Wooldridge, 1957). It was herniation between these parts of the diaphragm that was described, or rather postulated, by Morgagni (1760). Many hernial openings in this part of the diaphragm are, however, situated in the midline behind the

xiphisternum, with the part of the diaphragm normally attached to the sternum and to adjoining costal cartilage on each side being missing; and it seems clear that these are congenital defects. Such

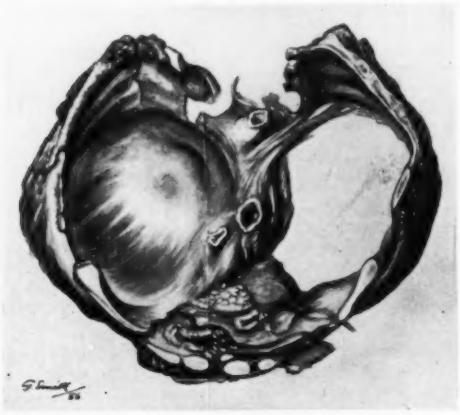


FIG. 2.—Drawing of an extensive defect of the left hemidiaphragm from an infant who died shortly after birth.

a defect is illustrated in *Fig.* 1. It is possible that some hernial openings in this region situated, not in the midline but to one or other side, are also congenital defects, though in this type of case, especially if discovered late in life, it must often be difficult to decide whether the condition is congenital or acquired. Dunhill (1935) and Holsby and Wells (1954) each described a retrosternal defect situated directly behind the xiphisternum; and Chin and Duchesne (1955) found that in 4 out of 6 retrosternal hernias operated on the defect extended across the midline. The 3 patients out of the series reported in this paper all had midline defects. One was a comparatively narrow defect little wider than the xiphisternum, and two were larger defects extending for some distance behind the costal cartilages on each side. In none of them was there any defect of the xiphisternum, an abnormality which Chin and Duchesne described as being present in some of their cases.

Congenital retrosternal hernias almost invariably extend into the anterior mediastinum and these probably always possess a hernial sac. Even when the opening in the diaphragm is in the midline, the presence of the heart on the left side causes the hernial sac to project towards the right. It was noted at operation on the three patients whose cases are described that the muscle of the diaphragm ended abruptly with a thickened edge at the margin of the defect, the hernial sac being of peritoneum only. Rarely, a defect of pericardium has been found associated with a congenital retrosternal diaphragmatic defect, abdominal viscera then herniating into the pericardial cavity (Gross, 1946; Wilson, Rumel, and Ross, 1947).

Extensive Defects of the Hemidiaphragm.—These are often referred to as absence of the hemidiaphragm, but as shown in *Fig.* 2 (a drawing of a specimen kindly supplied by Dr. J. E. Morrison from an infant who died shortly after birth, with most of the abdominal viscera in the left pleural cavity) the whole hemidiaphragm may not be absent. The left crus is present and in front there is a shelf of muscle lying behind the costal cartilages and the anterior ends of the ribs. It is somewhat tempting to try to regard this type of defect as being an unusually large defect of the posterolateral type. However, the difference between these very extensive defects and the small, or comparatively small, defects in the posterolateral region is so great that it seems preferable to regard them as a distinct type.

Hernias through these different defects vary in their clinical features and in the problems they present. They will be considered separately.

1. POSTEROLATERAL HERNIAS

Clinical Features.—

Urgency in the Newborn.—It has frequently been pointed out that this type of diaphragmatic hernia is often rapidly fatal, but it still does not seem to be realized as widely as it should be how many infants born with this type of congenital hernia die very soon after birth, almost always without the condition being diagnosed or even suspected. Morrison (1952), from post-mortem examinations of infants dying in Belfast maternity hospitals, found that diaphragmatic hernias were responsible for 1 in every 50 deaths in the first three days of life. He has told me that he found this the commonest congenital anomaly to cause death at this time. There were altogether 9 neonatal deaths from this cause. Six of these infants died within two hours of birth, two others within twenty-four hours, and the ninth lived for

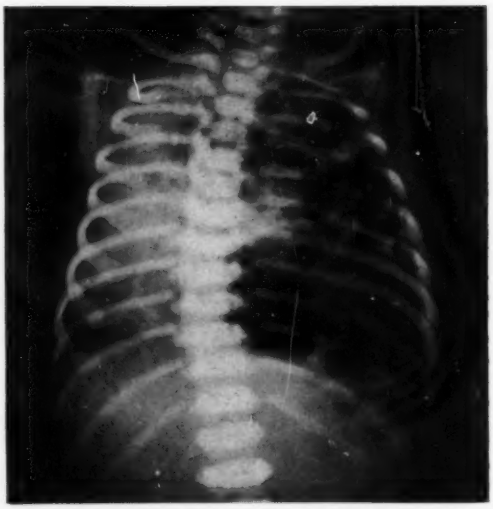


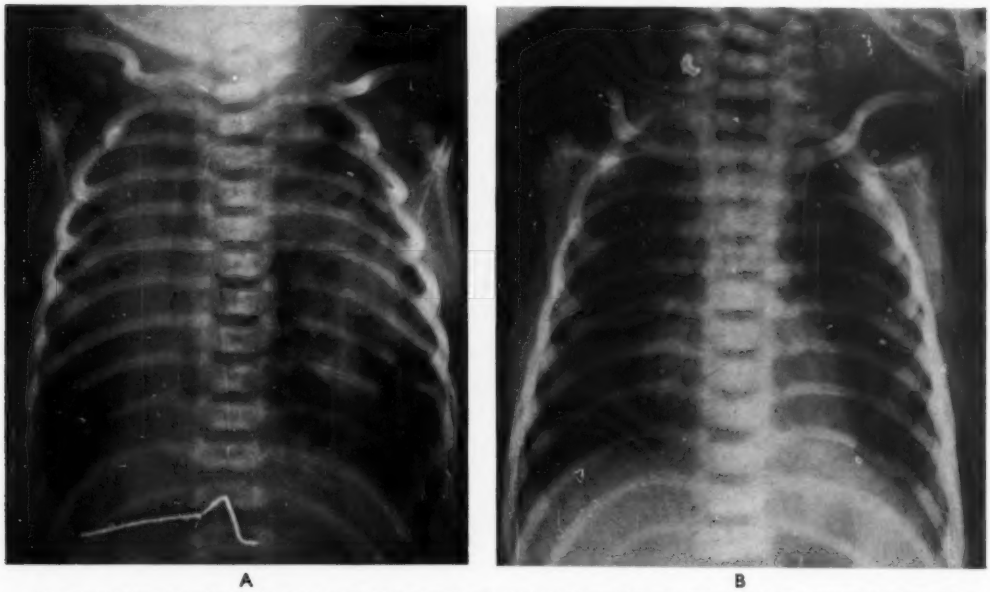
FIG. 3.—Radiograph of a 10-hour-old infant with a left-sided posterolateral diaphragmatic hernia (*Case* 2, *Table I*). The viscera herniated into the left chest have displaced the heart and mediastinum so much that the right lung is virtually completely collapsed. Note the almost complete absence of gas-containing bowel from the abdomen.

eight days. During this period no example of the condition was diagnosed in these hospitals during life, and without post-mortem examination the importance of this condition as a cause of death in

the newborn would not have been realized. It is clear that children with this condition who survive for even a few days, let alone for weeks, months, or years, are the exceptions.

The radiograph of *Case* 2 (*Fig.* 3) reveals clearly why these infants die so rapidly. The herniated viscera in the left chest have pushed the heart and mediastinum so far to the right that scarcely any aerated lung is seen even on this side. Death from anoxia is therefore certain to occur before long.

The condition should be suspected if an infant, in spite of making efforts to breathe, remains



A B

FIG. 4.—Radiographs of an 11-day-old infant with a left-sided posterolateral hernia (*Case* 9, *Table I*). A, The herniated viscera have again displaced the heart and mediastinum to the right. B, Radiograph 12 hours after operation showing the almost complete expansion of the left lung that can often be obtained at the time of operation.

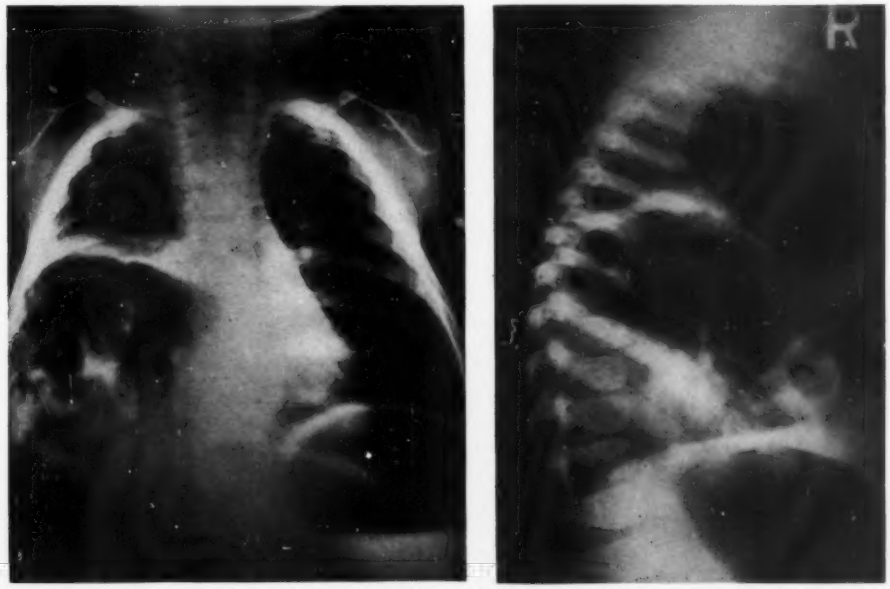


FIG. 5.—Radiographs of a 3-month-old girl with a right-sided posterolateral hernia with a comparatively small sac (*Case* 3, *Table I*)

dyspnœic, cyanosed, and distressed. Displacement of the heart-beat, usually to the right chest (this, incidentally, may be diagnosed mistakenly as 'dextrocardia'), and absence of breath-sounds on the left side would make one's suspicions stronger. Intestinal sounds may sometimes be heard in the chest, but this is not common. A chest radiograph should be taken on suspicion and will at once make the position clear.

Many of these infants die so rapidly after birth that there must often be virtually no chance of diagnosing the condition and having them operated on in time. But more lives could clearly be saved if those dealing with infants just after birth had the condition more in mind, and if it were customary to take chest radiographs of newborn infants suffering from respiratory embarrassment. Even if their condition is very critical, immediate operation offers

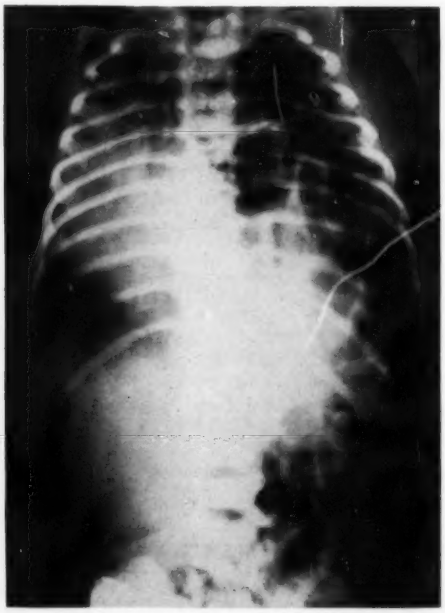


FIG. 6.—Radiograph of a 3-day-old boy with a left-sided posterolateral hernia with a large sac (*Case* 8, *Table I*). The herniated viscera do not extend to the apex of the chest, an appearance which usually, though not invariably, indicates that a sac is present.

an excellent chance of their survival. Operation should never be delayed in the hope that the condition of such infants will improve. This practice inevitably results in an exceedingly high mortality-rate.

When a comparatively small hernial sac limits the amount of viscera in the chest symptoms are naturally less severe. It is possible, however, as in *Case* 8, for a sac to be present, but to be so large that symptoms are just as severe as if no sac were present.

Symptoms in Older Infants and Children.—In those infants who survive the neonatal period the commonest symptom again is dyspnœa, associated at times with cyanosis. Failure to thrive is not uncommon, either because of poor feeding as a result of dyspnœa or because of vomiting.

Less often, vomiting is the main symptom. This was so in only one of my cases, *Case* 5, a girl aged 3 years, whose stomach was herniated into the left chest.

It is unusual for obstruction or strangulation to be caused in these cases by the intestines being constricted as they pass through the opening in the diaphragm, but this has occasionally been described (Cooke and Corner, 1949; Rickham, 1955). It did not develop in any of the cases in the present series.

Occasionally, the condition is detected by chance on a routine chest radiograph, there having apparently been no symptoms. One of my cases was picked up in this way, *Case* 7, a boy aged 12 years, in whom the small bowel alone had herniated. Though no symptoms had been noticed before operation, the parents realized after operation that the boy's exercise tolerance had been increased and that before this he had, in fact, been unduly dyspnœic on exertion.

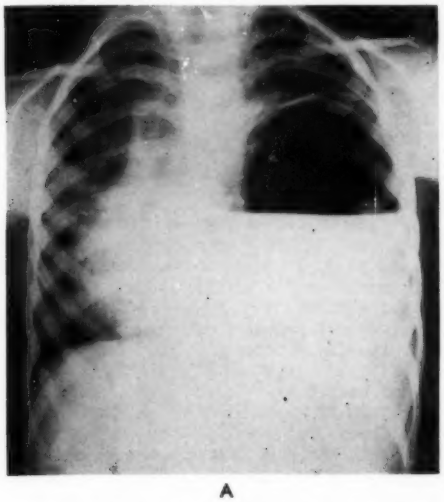
Radiological Findings.—*Figs.* 3 and 4 A, from *Cases* 2 and 9, show the typical radiographic appearance, gas-containing viscera being present in the left chest, and the mediastinum being displaced to the right. There is no necessity in such cases to do barium pictures; the appearance of the straight radiograph is diagnostic.

The postero-anterior and lateral views in *Fig.* 5, from *Case* 3, show clearly how a hernial sac can limit the amount of viscera that herniate into the chest. In *Case* 8, however, the hernial sac that was present was so large that not only were symptoms acute, but it was not recognized from the radiograph (*Fig.* 6) that a hernial sac was present. It can be seen that the herniated viscera did not extend to the apex of the chest, and it has been stated (Gross, 1953) that this appearance indicates that there is a hernial sac. This, however, is not invariably so. I have seen a similar appearance in one infant with free pleuroperitoneal communication (*Case* 10). In two older children also, *Cases* 5 and 7, the viscera did not extend to the apex of the chest, even though no hernial sac was present.

The radiographs of these two older children were rather unusual. *Fig.* 7 A, B, from *Case* 5, shows a distended stomach occupying a large part of the left chest. The appearance resembles that of an eventration of the diaphragm but the fundus of the distended stomach is higher in the chest than it would be with an eventration. A lateral radiograph after giving barium (*Fig.* 7 C) showed that the stomach and some of the intestines were passing up rather posteriorly and were therefore passing through a hernial defect. There was no hernial sac.

It was not certain at first whether the radiographs shown in *Fig.* 8 A, B, from *Case* 7, indicated a diaphragmatic hernia or not, but after a pneumoperitoneum had been induced another chest radiograph showed that a shallow pneumothorax was present, thus indicating free communication between peritoneal and pleural cavities through a diaphragmatic defect. The unusual appearance on the straight radiograph was due to small intestine only being herniated. This was made clear by barium meal (*Fig.* 8 C) and confirmed at operation.

Evolution of Surgical Treatment.—The technique now used for operating on these cases and the details of post-operative management were evolved largely as a result of the three cases in the series that ended fatally. These cases will therefore be described in more detail than is given in *Table I*. *Cases* 1 and 2 died from what were avoidable causes, if these had been known about at the time, and from both valuable lessons were learnt.



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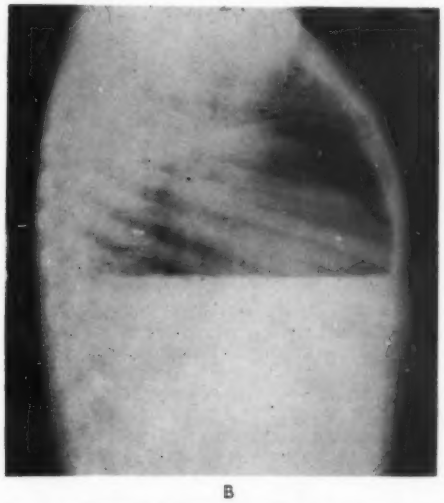


B

C

FIG. 7.—Radiographs of a 3-year-old girl with stomach herniated through a left-sided posterolateral defect (*Case* 5, *Table I*). A, B, Straight radiographs showing the stomach distended and occupying much of the left side of the chest. C, Barium meal showing stomach and intestine passing into the chest through a defect situated towards the posterior part of the diaphragm, the stomach at this time not being obstructed.

Case 1 was perhaps the most instructive case of all. This was a little boy, aged 4 months, who on Oct. 25, 1950, was transferred from Armagh Hospital to the Royal Belfast Hospital for Sick Children. He had always been rather poorly, with some dyspnœa and cyanosis. Six weeks before his transfer to Belfast he had started vomiting and losing weight. He was a thin, pale, undernourished infant, and dyspnœa was present at rest. There were no breath-sounds on the left side of the chest, and the heart impulse was palpable to the right of the sternum. Shortly after admission he became very ill, with pyrexia, cyanosis, and extreme dyspnœa. A radiograph showed the typical appearance, the left chest being occupied by gas-containing bowel and the mediastinum displaced to the right.

At operation on Nov. 12, 1950, the left chest was entered through the eighth intercostal space. The chest contained small intestine, cæcum, ascending and transverse colon. These had passed through a small defect in the posterolateral part of the diaphragm. The herniated viscera were replaced in the abdomen through the defect. This was a rather tedious and difficult procedure. The diaphragmatic defect was then closed by interrupted linen stitches. It was not particularly easy to place these stitches in the posterolateral corner of the diaphragm through an incision some distance higher in the chest. The chest was closed without drainage.

The child started vomiting several hours after operation and this continued. The stomach was aspirated and blackish fluid was withdrawn. Parenteral fluid was given, but the child died twenty-four hours after operation.

At post-mortem examination the stomach and proximal part of the duodenum were found to be grossly distended, and the second part of the duodenum to be obstructed by a band of peritoneum

passing over it from the hepatic flexure. The small intestine, cæcum, and ascending colon were quite free of attachment to the posterior abdominal wall. This had allowed the proximal colon to be displaced towards the left, so that the peritoneal band was pulled tightly across the duodenum. The abnormality was not a malrotation, but lack of attachment to the posterior abdominal wall of a normally rotated cæcum, ascending colon, and small intestine.

It was clear that this fatality had been the result of non-fixation of the midgut; and it also seemed clear that this abnormality must be present in most congenital hernias of this type, for otherwise how could the whole of the small intestine, the cæcum, and the ascending colon herniate into the chest? And, in fact, out of the eleven posterolateral cases in the present series there was non-fixation of the midgut in 8. In one of these cases there was, in addition, incomplete rotation of the colon, the cæcum in this case lying close to the duodenum. Because of this high incidence of abnormalities of rotation and fixation of the midgut it would seem reasonable to expect that duodenal obstruction would be a not uncommon complication in these cases.

It is surprising, therefore, to find scarcely any reference in the literature on diaphragmatic hernias to abnormalities of midgut attachment ever causing any trouble. It seems clear, though, that they do cause trouble, and not only after operation. That this is so finds some support from the present series, for the two patients who were more than a few months old had the midgut normally attached, whereas the more usual finding of non-attached midgut was present in 8 out of the 9 small infants. Vomiting, as already mentioned, is not uncommon in children with this type of hernia, and Harrington (1942) has pointed out that in these children the stomach may be markedly dilated, even in cases in which it remains in the abdomen. He did not put forward any explanation for this gastric dilatation, but the most likely explanation would seem to be that it is the result of duodenal obstruction. It would, indeed, be surprising if this condition did not develop in these cases before operation as well as after reduction of the bowel into the abdomen, and in the case just reported the vomiting before operation, like that after operation, may well have been caused in this way. A more complete volvulus of the midgut leading to intestinal obstruction or strangulation may, of course, also result from abnormalities of rotation and fixation of the midgut; and one of the infants with posterolateral diaphragmatic hernias reported by Moore, Battersby, Roggenkamp, and

Table I.—Details of Cases of Congenital Posterolateral Diaphragmatic Hernia

Case No.	Age and Sex	Symptoms	Type of Hernia	Viscera Herniated	Attachment of Midgut	Operation	Result
1	4 mth. M.	Dyspnœa, cyanosis, vomiting	L. side No sac	Small intestine, cæcum, ascending and transverse colon	Not attached	Thoracic approach, reduction of viscera, suture of defect	Died in 48 hr. from duodenal obstruction
2	12 hr. F.	Dyspnœa, cyanosis	L. side No sac	Stomach, spleen, small intestine, cæcum, colon	Not attached	Abdomino-thoracic approach, reduction of viscera, Ladd's procedure, suture of defect	Died in 1 hr. from tension pneumothorax
3	3 mth. F.	Recurrent respiratory infections	R. side Sac present	Part of liver, small intestine, cæcum, colon	Not attached	Abdomino-thoracic approach, reduction of contents, excision of sac, Ladd's procedure, suture of defect	Cured
4	4 mth. M.	Dyspnœa, cyanosis	L. side No sac	Small intestine, cæcum, colon	Not attached	Abdomino-thoracic approach, reduction of viscera, Ladd's procedure, suture of defect	Cured
5	3 yr. F.	Dyspnœa, vomiting	L. side No sac	Stomach, spleen, some intestine	Attached	Abdomino-thoracic approach, reduction of stomach and intestine, suture of defect	Cured
6	6 d. F.	Dyspnœa, cyanosis	L. side No sac	Small intestine, cæcum, colon	Not attached	Abdomino-thoracic approach, reduction of viscera, Ladd's procedure, suture of defect	Cured
7	12 yr. M.	Slight dyspnœa on exertion	L. side No sac	Small intestine	Attached	Abdomino-thoracic approach, reduction of intestine, repair of defect with nylon patch	Cured
8	3 d. M.	Dyspnœa, cyanosis	L. side Sac present	Stomach, spleen, small intestine, cæcum, colon	Not attached	Abdomino-thoracic apporach, reduction of viscera, Ladd's procedure, excision of sac, suture of defect	Died 3rd day from tracheobronchial secretions causing bronchial obstruction
9	11 d. F.	Dyspnœa, cyanosis	L. side No sac	Small intestine, cæcum, colon	Not attached Malrotated	Abdomino-thoracic approach, reduction of viscera, Ladd's procedure, suture of defect	Cured
10	3 wk. M.	Dyspnœa, cyanosis	L. side No sac	Small intestine, transverse colon	Attached	Abdomino-thoracic approach, reduction of viscera, suture of defect	Cured
11	2 wk. M.	Dyspnœa, cyanosis	L. sac No sac	Spleen, small intestine, cæcum, colon	Not attached	Abdomino-thoracic approach, reduction of viscera, Ladd's procedure, suture of defect, chest drain to water seal	Cured

Campbell (1957) died from midgut volvulus five weeks after operation. It is impossible to estimate how frequently these two conditions complicate diaphragmatic hernias of this type, either before or after operation, for in many of the reported series no details are given of cases that ended fatally. In any case, there is always the possibility that these conditions may develop a considerable time after operation.

Following the unfortunate outcome in this first case of the series I decided that when operating again on such a case I would take steps, when non-fixation of the midgut was present, to prevent either duodenal obstruction or midgut volvulus from developing. This can be achieved quite simply by the operation described by Ladd (1933) for dealing with malrotation or non-fixation of the midgut, namely, by division of the peritoneal fold that passes across the duodenum and fixes the hepatic flexure or incompletely rotated cæcum to the posterior abdominal wall. This procedure cannot be carried out by means of a purely thoracic approach. A purely abdominal approach would certainly allow this, but seemed to have certain disadvantages. I feared that at times it might be difficult to reduce the herniated viscera, particularly, perhaps, on the right side, for the liver when

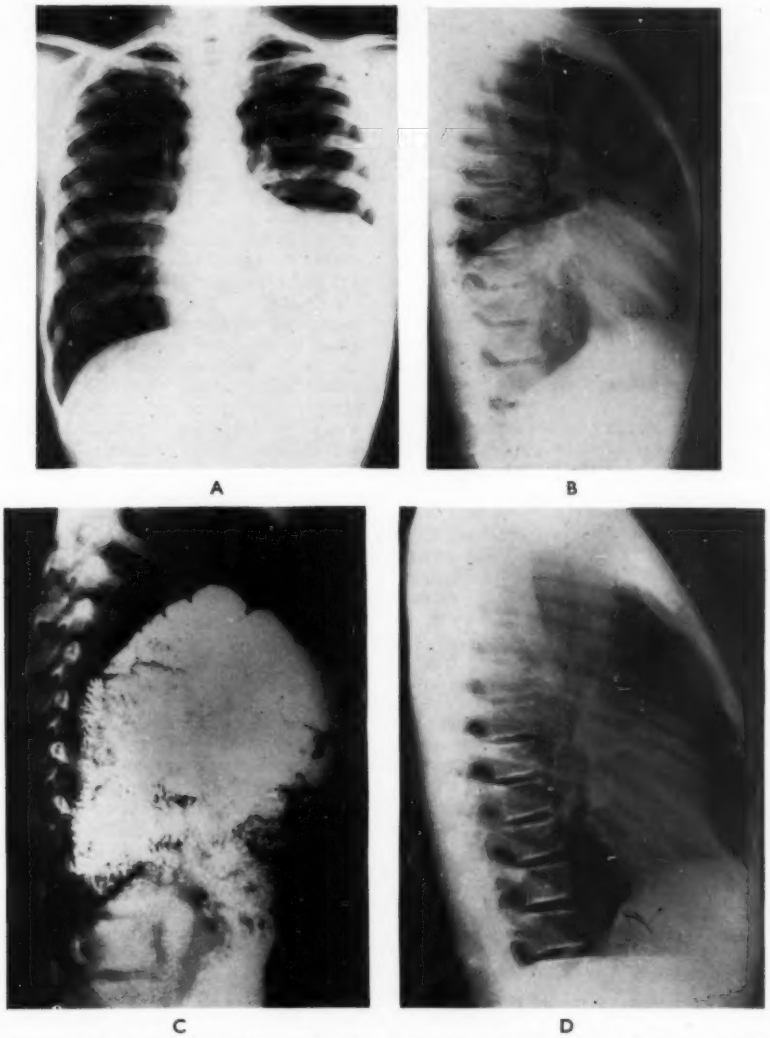


FIG. 8.—Radiographs of a 12-year-old boy with small intestine herniated through a posterolateral defect (*Case* 7, *Table I*). A, B, Straight radiographs showing radio-opaque mass in lower part of left chest. C, Barium meal showing small intestine passing into the chest through a diaphragmatic defect. D, Radiograph two years after repair of the defect with a patch of nylon fabric.

partly herniated tends to become somewhat dumb-bell shaped, the constriction being at the hernial orifice. And I thought that it might also, perhaps, prove rather difficult to suture the diaphragmatic defect through an abdominal incision, particularly again on the right side, where the liver, after reduction into the abdomen, might tend to be in the way. These fears about the difficulties of a purely abdominal approach were very likely exaggerated; but, in any case, because of them I decided to adopt a thoraco-abdominal approach, entering the thorax low down posterolaterally between the tenth and eleventh ribs and passing across the abdomen towards the umbilicus. I have used this approach in all cases since then.

When *Case* 2 presented, the changes in operative technique decided on as a result of the previous case were put into effect. This was a female infant who, on Jan. 27, 1952, was transferred from the Royal Maternity Hospital, Belfast, to the Royal Belfast Hospital for Sick Children when 12 hours old. She had been dyspnœic and cyanosed from birth and on admission was comatose and virtually moribund. A chest radiograph (*Fig.* 3) taken before transfer had made the diagnosis clear and immediate operation was carried out.

At operation only oxygen was administered to begin with. A left thoraco-abdominal approach, as outlined above, was used. When the viscera were delivered from the chest it was seen that the heart was almost at a standstill, beating at perhaps about 40 beats a minute. However, within a minute of the lungs being better inflated the heart began beating strongly and rapidly, and the child's condition remained perfectly satisfactory from then onwards. There was, as expected, complete non-fixation of the midgut and Ladd's procedure was carried out. In spite of every effort to inflate the left lung fully, it could not be expanded to occupy more than about the upper third of the chest. The small defect in the diaphragm was closed and the chest and abdomen closed without drainage. The child's condition was good at the end of the operation, but an hour later she died quite suddenly.

At post-mortem examination the cause of death was found to have been a tension pneumothorax on the left side. There had been some alveolar rupture leading to interstitial emphysema and finally to rupture into the pleura and the development of a tension pneumothorax.

It was learned from this unfortunate fatality that too strenuous efforts must not be made by the anæsthetist to get the hitherto collapsed lung to occupy the whole of the pleural cavity on the affected side. It may be that previously unexpanded and possibly incompletely developed lung can be ruptured even more readily than normal lung. Since then, care has been taken to avoid excessive pressure in attempts to expand the lung, though in point of fact, in all cases since then except one (*Case* 11) the lung has expanded reasonably easily to occupy at least the greater part of the pleural cavity, as is shown in *Fig.* 4 B.

The cause of death in the third fatal case (*Case* 8)—obstruction of the air passages by tracheo-bronchial secretions—probably remains as the chief hazard post-operatively in young infants. This patient was a male infant who, on Jan. 24, 1957, was admitted to the Royal Belfast Hospital for Sick Children, three days after a normal home birth, because of dyspnœa and cyanosis. A chest radiograph showed bowel in the left hemithorax extending almost to the apex of the chest and pushing the mediastinum over to the right (*Fig.* 6).

At operation, on the day of admission, it was found that there was a large hernial sac arising from the periphery of the diaphragm, close to its attachment to the tenth and eleventh ribs. This contained stomach, spleen, small intestine, cæcum, and ascending colon, the sac being so large that it was scarcely limiting in any way the amount of viscera herniated into the thorax. The operation was carried out in the way outlined above and described in more detail later. There was, once again, non-fixation of the midgut, and the Ladd procedure was again carried out. The sac was excised, the defect in the diaphragm sutured, and the chest and abdomen closed.

Probably from over-confidence as a result of several successful cases, less attention than usual was given in this case to post-operative care. A surgical incubator was not used, as it had been in earlier infant cases, and when the intravenous infusion stopped soon after the operation was completed this was not set up again. Instead, oral feeds were started within twenty-four hours. The infant's condition gave rise to no anxiety for two days after operation. His condition had seemed satisfactory early on the second day, but in the evening, about forty-eight hours after operation, it was noted that he was cyanosed and dyspnœic. It was not until several hours later, however, during the night, that it was realized that the child was acutely ill, and very soon after this he died.

At post-mortem it was found that the major bronchi contained a moderate amount of thick mucus, with the mucosa slightly congested. There were scattered areas of atelectasis in the right lung and complete atelectasis of the left lung. A chest radiograph on the day after operation had shown quite satisfactory expansion of the left lung and no abnormality of the right lung.

One of the chief post-operative hazards in neonatal surgery is that an infant's tracheo-bronchial tree is so easily obstructed by mucoid secretion, and this danger is perhaps greater after operations for these congenital diaphragmatic hernias than for most other conditions. After the herniated viscera have been returned to the abdomen and the diaphragm and abdominal wall closed, the abdomen, not having previously accommodated these viscera, is tight and tense. The child's respiration, therefore, tends to be embarrassed by restricted diaphragmatic movement, and there must consequently be an increased tendency both for bronchial secretions to be retained and for any degree of bronchial blockage to produce dangerous anoxia. While greater post-operative attention in this case might not, of course, have led to any different result, the case did at least emphasize the importance of post-operative care, especially in the case of infants. The greatest attention is now paid to this.

Surgical Technique and Management.—The following plan of treatment has been arrived at and is now used routinely.

Pre-operative Preparation.—Little of this is required. Normally, an intravenous infusion is set up, only glucose being used in infants. The chief advantage in having an intravenous infusion going is that the anæsthetist can use it to give injections of muscle relaxants, and in addition blood-loss may be replaced. But in the case of an infant *in extremis*, time should not be wasted putting up a drip if this would mean any delay in operating. Relaxants in such cases can be given subcutaneously with hyalase.

FIG. 9.—The position of the skin incision in the operation for a left-sided congenital posterolateral hernia, and the appearance after opening the chest and abdomen. The incision into the abdomen has not yet been connected with the opening in the diaphragm. This cannot be seen as it is hidden by the coils of bowel.

FIG. 11.—Appearance after division of the transduodenal band and mobilization of the colon so as to expose the superior mesenteric artery.

Anæsthesia.—This is as for any operation involving thoracotomy. An intratracheal tube should always be passed, for in infants particularly, attempts to inflate the lungs using a face-mask result in the stomach becoming distended, and this should definitely be avoided. Details of anæsthesia will not be gone into, but one might point out that in young infants hypnosis is best maintained with nitrous oxide and oxygen, supplemented at times with fluothane, and relaxation with injections of suxamethonium ('scoline'). In older children the anæsthetic is conducted on the same lines as in adults.

Technique of Operation.—As far as the general conduct of the operation is concerned, the only special point again concerns young infants. It is important to avoid chilling of these patients during operation. This may be achieved most conveniently by placing the infant on a heated surface, such as a partly filled, well padded, hot-water bottle. The patient is placed in the right or left lateral position as required. In infants this position is readily maintained without any special fittings.

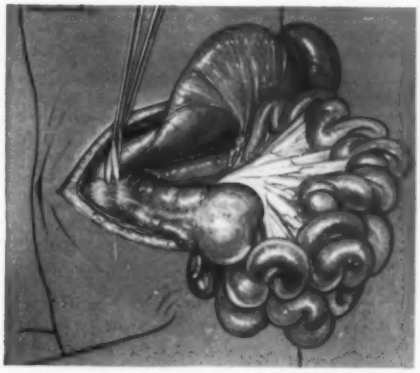


FIG. 10.—After withdrawing the viscera from the chest non-fixation of the midgut has been found. The peritoneal band passing across the duodenum from the hepatic flexure is being divided with scissors (the Ladd procedure).

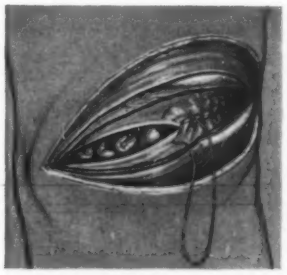


FIG. 12.—The viscera have been returned to the abdomen and the diaphragmatic defect and abdominal wall are being sutured.

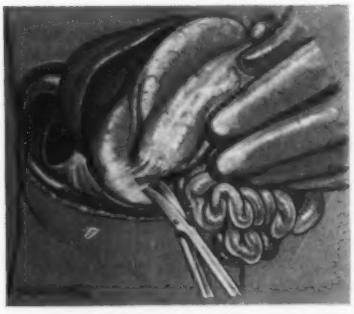


FIG. 13.—The Ladd procedure being carried out during an operation for a right-sided posterolateral hernia. The diaphragmatic defect can be seen.

The steps in the operation are illustrated in *Figs.* 9–13. The incision commences posteriorly

between the tenth and eleventh ribs and extends forwards towards the umbilicus, reaching as far as the edge of the rectus sheath. The posterior part of the incision is deepened so as to open into the pleural cavity. Care must be taken that as the pleura is incised underlying viscera are not damaged, but once a small opening in the pleura has been made this is safely and quickly enlarged with scissors. Attention is now turned to the abdominal part of the incision, and the peritoneal cavity is opened from the edge of the rectus sheath to the attachment of the diaphragm to the tenth and eleventh ribs. One can now see that the defect in the diaphragm lies immediately under the incision, right at the periphery of the diaphragm in the region of its attachment to these ribs. It is best at this stage to join up the incision in the abdominal wall with the defect in the diaphragm, for this step removes any possible difficulty in withdrawing the herniated viscera from the chest and, in addition, probably makes it easier at a later stage to suture the defect in the diaphragm. There is, of course, no costal margin to cut through at this level. Once the abdominal viscera have been withdrawn from the chest any difficulty the anæsthetist may have had in maintaining proper oxygenation disappears and any necessity for hurry is over. The operation can proceed calmly and deliberately.

The viscera are now arranged in as normal a position as possible and the attachment of the ascending colon and hepatic flexure is examined. In the majority of cases the cæcum and ascending colon, and also the small intestine, will be found to be free of attachment to the posterior abdominal wall, and the hepatic flexure will be found to be attached by a band of peritoneum passing across the second part of the duodenum. One has only to lift the ascending colon gently towards the left to see how readily duodenal obstruction could be produced. And, of course, volvulus of the whole midgut could also easily be brought about. Less often, there is malrotation of the bowel, the cæcum being attached in the region of the duodenum, but the mesentery of the small intestine not being attached.

The simplest method of dealing with these abnormalities, and of avoiding any possibility of duodenal obstruction or of midgut volvulus developing later, is by means of the procedure described by Ladd (1933). The cæcum and ascending colon are drawn towards the left and the peritoneal band passing across the duodenum is divided with fine scissors. With a little gentle blunt dissection and division of a few fine strands with scissors the duodenum is cleared as far as the superior mesenteric vessels. The position now is that the small intestine is attached to the posterior abdominal wall at its commencement at the duodenojejunal flexure and the ascending and transverse colon at the splenic flexure. As a result, the cæcum and ascending colon can now shift to the left without any tendency to produce either duodenal obstruction or volvulus.

It is clear from the literature that, as a rule, no attempt has been made during operations on diaphragmatic hernias of this type to carry out any procedure aimed at preventing the complications that may arise from abnormal attachment or rotation of the midgut. Harrington (1942) advised that care should be taken when replacing the intestines in the abdomen not to produce a volvulus; Riker (1954) suggested that it might be well to look for and divide any abnormal bands that might later produce obstruction; but the only reference I have found to steps similar to those described in this paper actually being taken during operation is by Gross (1953). He states that in his department such steps have been carried out in 12 out of a total of 72 diaphragmatic hernias, these including 63 posterolateral hernias. It was somewhat difficult at first to see why the proportion of cases treated in this way should be so much higher in the present series. However, Gross (1958) has pointed out that in the Boston Children's Hospital it is not the practice invariably to carry out Ladd's procedure in those cases in which the bowel is not properly attached, a decision on whether this step is carried out or not in such cases depending to some extent on the experience of the particular operator in each case. Even though many cases with abnormalities of midgut attachment clearly escape any trouble, at least in the immediate postoperative period, obstruction of the duodenum and volvulus of the midgut are definite dangers, and as the carrying out of the Ladd procedure adds but a few minutes to the operation, I feel that in such cases this step should certainly form part of the operative technique. In none of the seven cases of the present series in which the Ladd procedure was carried out was there any reason to regret having done so.

It only remains to close the defect in the diaphragm and the incision in the abdominal and thoracic wall. Sometimes the attachment of the splenic flexure of the colon extends on to the margin of the defect and requires to be separated a little so that suturing of the defect may be carried out without any danger of damaging the bowel. There is no necessity deliberately to denude the edge of the defect of its serous covering, but if a hernial sac has been present then, of course, the cutting away of the sac provides a denuded edge. Starting at the part of the defect farthest removed from the rib cage, it is usually possible, with a continuous suture, to bring the edges of the defect together and then to continue this same suture to the anterior end of the abdominal incision, closing with it the transversus abdominis muscle and peritoneum. Non-absorbable material, such as linen, is used for this layer. Approximating the tenth and eleventh ribs with finger and thumb as each stitch in the diaphragm is tightened lessens the risk of these stitches cutting through.

Next, the anæsthetist inflates the lung, being particularly careful not to use excessive pressure, and the thorax is closed by approximating the tenth and eleventh ribs with two pericostal catgut sutures. If the previously collapsed lung does not expand readily, the anæsthetist should first make sure, by withdrawing the intratracheal tube a little and inflating again, that this is not due to the tube being in the opposite bronchus. If it should still not prove possible to get the lung satisfactorily inflated, then before the chest is closed it is probably advisable to introduce a small tube through an intercostal stab wound and connect it with a water-seal bottle. This will allow expansion of lung to occur as rapidly as possible. As mentioned earlier, however, the lung has usually been inflated by the anæsthetist, without

difficulty, to occupy at least the greater part of the pleural cavity, and in only one case (*Case* 11) was a chest drain used. Twenty-four hours after operation a radiograph showed that the lung was occupying most of the pleural cavity and the drain was removed. After approximating the ribs the internal and external oblique layers in the abdominal wall are sutured with continuous catgut, the external oblique suture being continued into the lower part of the latissimus dorsi muscle on the chest wall, and the skin is closed.

As already pointed out, closure of the abdomen is, as a rule, obtained only with some tension, for the abdominal cavity, not having until now contained the previously herniated viscera, is smaller than it would normally be. I have always been able to close all layers of the abdominal wall, however, and usually without much difficulty, and have not had to resort to the manœuvre, at one time recommended by Ladd and Gross (1941) as being useful at times, of closing only the subcutaneous tissue and skin, and then reopening the wound and suturing all layers several days later. The use of muscle relaxants undoubtedly makes closure easier, but, in addition, one of the advantages of the almost transverse incision in the abdominal wall is that the first layer to be sutured, the transversus abdominis and peritoneum, holds stitches extremely well. Two steps which are of help in obtaining closure in difficult cases are, first, having the anæsthetist pass a catheter into the stomach so that it can be deflated, and, secondly, stretching the abdominal wall, with the aim of enlarging the peritoneal cavity. This rather tense closure of the abdomen is the one step in the operation that in infants appears at times to produce some degree of shock. It is now, just as the operation is finishing, that they have sometimes become rather blanched and shocked looking. This, however, has always passed off quickly and has not required any special treatment, or, indeed, been enough to give rise to any particular anxiety.

In some cases the actual closure of the defect in the diaphragm is not quite so readily carried out as has been described above. Sometimes the outer margin of the defect is so flush against the tenth or eleventh ribs that no tissue can be found at this point through which to pass a needle. However, separating the diaphragm and transversus abdominis muscle from their attachment for a short distance beyond the defect readily provides sufficient tissue for suturing. In only one of my cases (*Case* 7) was the defect so large that its edges could not be approximated. Possible extensive subperiosteal stripping of neighbouring ribs, and perhaps excision of ribs, would have allowed the defect to be closed by suturing. However, I preferred to suture a piece of nylon fabric in position as a patch, using interrupted linen stitches to fix it around the edge of the defect. This worked well and has given rise to no trouble. Radiographs taken two years after operation show a normal appearance (*Fig.* 8 D). In no case has the phrenic nerve been crushed, and in my opinion this should be avoided, for it must inevitably reduce respiratory efficiency. Indeed, paralysis of the diaphragm, at least on the right side, in an infant can cause serious respiratory distress (Bingham, 1954).

The thoraco-abdominal approach described above has proved extremely satisfactory. It is easily and rapidly made, the viscera can rapidly be delivered from the chest and the lungs inflated, the Ladd procedure can be carried out when indicated, and as the incision is directly over the defect in the diaphragm this can be closed with the least possible difficulty. There are, too, other advantages in having the thorax open as well as the abdomen. As was found in *Cases* 2 and 11, and as emphasized by Moore and others (1957), it is sometimes impossible to obtain good expansion of the collapsed lung on the side of the hernia, and it must be easier to observe through the intercostal incision how expansion of the lung is proceeding, than it would be by looking from the abdomen through a small defect in the diaphragm. When operating entirely from the abdomen it is, moreover, quite possible in cases with a large hernial sac, occupying a considerable part of the pleural cavity, to suture the defect in the diaphragm without recognizing that a hernial sac is present. This will then fill with fluid in the early post-operative period and interfere with expansion of the lung. Moore and others (1957) report that out of 11 cases of posterolateral diaphragmatic hernias in infants a hernial sac was overlooked in 3.

I feel sure that an operative approach similar to the one just described must have been employed by other surgeons when operating on diaphragmatic hernias of this type. Nevertheless, from a fairly extensive search of the literature I have not seen a description of just such an approach being used in these cases. Most surgeons have employed either an abdominal or a thoracic approach; a few have used separate abdominal and thoracic incisions, this being to facilitate reduction; and a few have employed an oblique incision in the epigastrium extending across the costal margin into the eighth intercostal space. This latter incision would provide some of the advantages of the incision described above, but not being directly over the defect in the diaphragm it would seem likely that herniated viscera could not be quite so easily reduced nor the defect quite so easily sutured.

Post-operative Care.—No special measures are required for older children, except that for a few days nothing more than sips of fluid should be given by mouth. This allows any tendency to intestinal distension to pass off and the rather tense abdomen to become soft. Parenteral fluids are continued during this time.

Small infants are best nursed in a surgical incubator with control of oxygen content, temperature, and humidity. A raised oxygen concentration (40 per cent) should be maintained for a few days in case there should be any tendency to anoxia, either from the tenseness of the abdomen causing respiratory embarrassment or from retained bronchial secretions. Humidity approaching 100 per cent prevents bronchial secretions becoming more viscid and thus reduces the danger of bronchial blockage and atelectasis. In these young infants, as in other cases, fluid intake by mouth is at first severely limited. This is a most important point in post-operative management, for some degree of intestinal ileus is probably inevitable, and the sooner this settles down and the tense abdomen becomes soft, the sooner does free diaphragmatic movement occur. There is, too, the danger of vomiting and of aspiration of vomited

material if feeds are started before this. If, because of the urgency of operation, an intravenous drip has not been set up beforehand, one should be set up at the end of the operation.

It is not very uncommon for some fluid to form in the chest post-operatively, for if the lung is slow in expanding to occupy the whole of the pleural cavity a negative pressure effusion naturally develops. This fluid disappears as further expansion of lung occurs. Aspiration has not been indicated in any of my cases.

2. RETROSTERNAL HERNIAS

Clinical Features.—Herniation through anterior or retrosternal diaphragmatic defects presents a completely different clinical problem from herniation through posterolateral defects. It would seem that symptoms of any kind, even minor symptoms, seldom occur during infancy or childhood, and most cases probably remain symptomless throughout life. Certainly more cases are discovered as incidental findings on a chest radiograph or at post-mortem examination after death from some other cause than

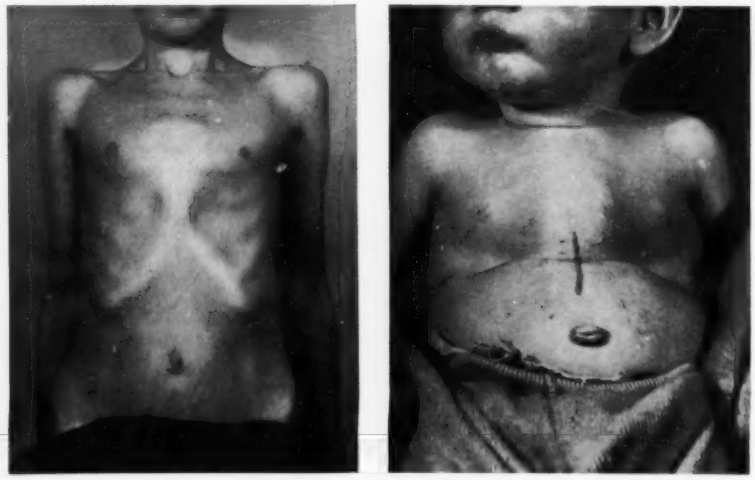


FIG. 14.—Photographs showing the projection forwards of the lower part of the sternum in two children with retrosternal diaphragmatic defects (*Cases* A and B, *Table II*).

Table II.—DETAILS OF CASES OF CONGENITAL RETROSTERNAL DIAPHRAGMATIC HERNIA

CASE NO.	AGE AND SEX	SYMPTOMS	TYPE OF DEFECT	VISCERA HERNIATED	OPERATION	RESULT
A	7 yr. F.	Chest deformity. Occasional abdominal pain perhaps due to hernia	Midline, narrow	Transverse colon and left lobe of liver	Epigastric incision, reduction of contents, suture of defect side-to-side	Cured
B	8 mth. M.	Chest deformity only	Midline, broad	Transverse colon only	Epigastric incision, reduction of contents, suture of posterior edge of defect to posterior rectus sheaths	Cured
C	72 yr. F.	Two attacks of sub-acute obstruction within 1 yr.	Midline, fairly broad	Transverse colon and stomach	Epigastric incision, reduction of contents and excision of sac, suture of posterior edge of defect to posterior rectus sheaths	Cured

Because of the danger associated with pulmonary infection if this should develop, antibiotics are used routinely after operation, combined therapy with penicillin and streptomycin being usually employed.

The only major complication post-operatively, apart from those that caused death in the three cases described in detail, occurred in *Case* 3. This little girl aged 3 months developed an ileo-ileal intussusception that was reduced by operation on the eighth day. Gross (1953) reports that he has had this condition develop post-operatively on two occasions.

present with symptoms. However, it sometimes happens, usually late in life, that obstructive symptoms develop. The main features of the present cases are shown in *Table II* and it can be seen that in *Case* C, a woman of 72 years, there had been two attacks of subacute obstruction during the year before operation. This complication is to be explained by the increasing fat deposits in mesentery and omentum that occur in some persons with increasing age. Because of the possibility of this complication occurring later in life it is probably a wise policy to operate on children or fit adults found

to have such hernias, even when they are quite symptomless and certainly if they contain any hollow viscera, especially as the condition can be dealt with by a comparatively simple operation.

A portion of transverse colon with its attached omentum is the viscus found most commonly in these retrosternal hernias, and as a rule this is the only hernial content. Sometimes, as in *Case* A, the left lobe of the liver also herniates through the defect, and sometimes, as in *Case* C, the stomach herniates along with the transverse colon. Small intestine has in a few reported cases also been found in these hernias, and sometimes, in addition, the cæcum and ascending colon, the midgut in these cases not being attached to the posterior abdominal wall (Dunhill, 1935; Harrington, 1942). As stated earlier, a peritoneal sac extending into the mediastinum is present in retrosternal hernias. This was so in each of my three cases.

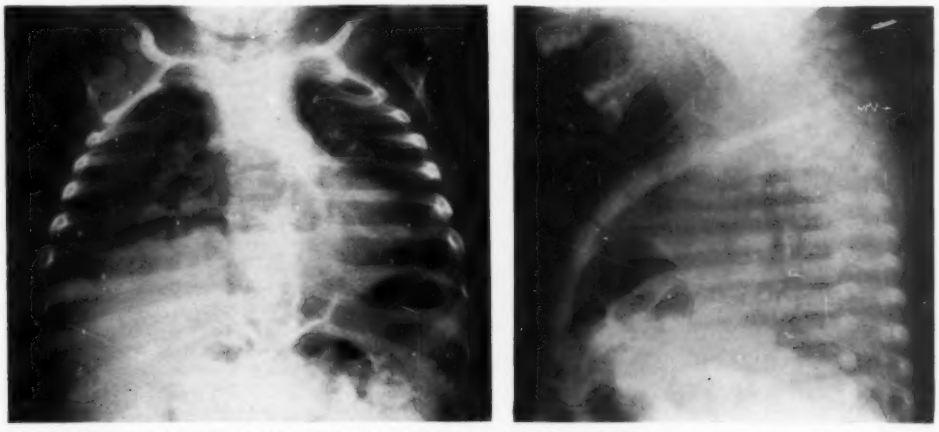


FIG. 15.—Radiographs of an 8-month-old boy with a loop of transverse colon herniated through a retrosternal defect (*Case* B, *Table II*). The gas-containing loop of bowel shows clearly on the straight radiographs.

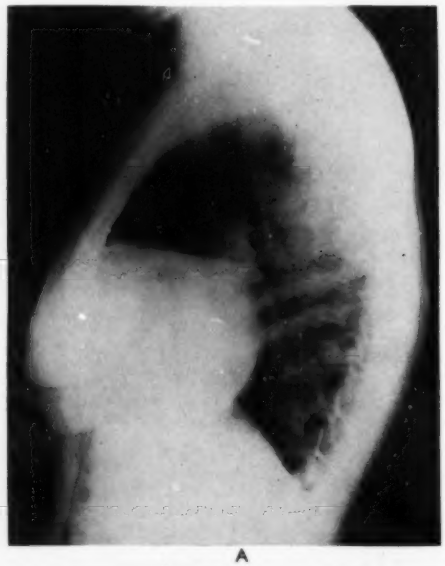
Association with Chest Deformity.—The two children with this type of diaphragmatic defect whom I have seen each had a chest deformity that I have not seen described elsewhere as being associated with this condition, but which could well be the result of the defect in the diaphragm. As shown in *Fig.* 14, the lower part of the sternum in both children projected forward beyond the ribs and costal cartilages. In the case of the younger child, indeed, this deformity was the reason for him being brought to hospital. A similar prominence of the sternum can be present to some extent without a retrosternal hernia being present; but the fact remains that this is exactly the deformity one might well expect to occur with the diaphragm attached to costal cartilages and ribs on each side and not attached to the sternum. It is, moreover, exactly the reverse of the funnel-chest deformity, which is possibly due to the sternum and costal cartilages being pulled backwards by an abnormal attachment of the diaphragm.

Radiological Findings.—A straight radiograph is sometimes sufficient to make the diagnosis. The postero-anterior and lateral radiographs of *Case* B (*Fig.* 15) show a loop of gas-containing colon behind the sternum. Barium meal and barium enema pictures will, of course, make the exact position clear. In *Case* C, it was demonstrated in this way that part of the stomach and the transverse colon had herniated (*Fig.* 16).

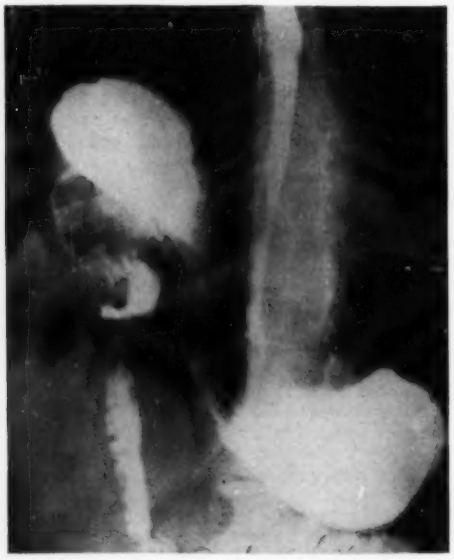
Surgical Treatment.—It is possible to deal with retrosternal hernias either through a thoracotomy incision or through an incision in the epigastrium. I have employed an epigastric approach in the three cases I have operated on and have found it very satisfactory.

A midline epigastric incision is made extending right up to the xiphisternum. On looking up beneath the xiphisternum the transverse colon with its omentum is seen passing through the retrosternal defect, and is as a rule easily delivered into the abdomen. So, too, are any other herniated viscera, such as a portion of liver or of stomach. In those cases in which the midgut is not attached in the abdomen, so that ileum, cæcum, and ascending colon herniate through the defect, I feel sure it would be wise at this stage to carry out Ladd's procedure. The peritoneal sac, if it can readily be withdrawn from the thorax, is excised, but this is not essential. In two of my three cases the sac was left behind and has caused no trouble. The falciform ligament is drawn into the sac, and when this is not removed the ligament requires to be divided before the edge of the defect can be defined. The method of closing the defect depends on its breadth. If it is narrow from side to side, as in *Case* A, it is most easily closed by suturing the edges together in the midline, starting at its most posterior point, coming forward to the xiphisternum, and continuing down the peritoneum and linea alba. If, however, the defect is broader, extending for a little distance behind the adjacent costal cartilages, as in *Cases* B and C, it is necessary to close the defect by bringing its posterior edge forward to the posterior rectus sheaths and xiphisternum, using interrupted mattress sutures of silk or linen. If the defect should be so large that it proved impossible or very difficult to close it in this

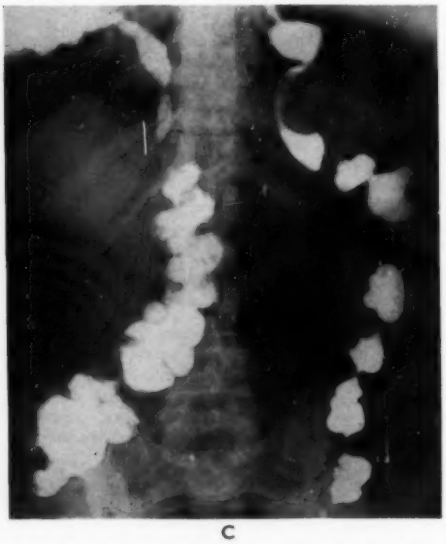
way, I should think that closure would be made easier by freeing the posterior rectus sheaths and linea alba from the xiphisternum and costal cartilages. I have not, however, had to do this in any of my three cases. Another method of dealing with a large defect, here as in the posterolateral region, would, of course, be to close it with a piece of nylon or other suitable material.



A



B



C

FIG. 16.—Radiographs of a 72-year-old woman with a retrosternal hernia (*Case* C, *Table II*). A, Lateral view, showing the anterior position of the hernia. The stomach is almost full of fluid. B, C, Barium meal and barium enema pictures, showing the distal part of the stomach and the transverse colon passing through the diaphragmatic defect.

3. HERNIAS WITH EXTENSIVE DEFECTS OF THE HEMIDIAPHRAGM

This type of diaphragmatic defect is very rare. The only example I have seen is the diaphragm illustrated in *Fig.* 2, from an infant who died shortly after birth. Most reported examples, indeed, have been from museum specimens or from autopsies on newborn infants, and it is clear that these defects are usually rapidly fatal. Clinically and radiologically such infants must be indistinguishable from infants with small posterolateral defects, but the only report I have seen of such extensive defects having been found at operations on infants is by Neville and Clowes (1954). In two infants with diaphragmatic hernias, one right-sided and the other left-sided, they found the hemidiaphragms almost completely absent. They were able to close the defects by suturing the right or left lobes of the liver to the chest wall and to the margin of the diaphragmatic defect. One of these infants lived; the other died from cardiac arrest at the end of the operation.

Non-fixation of the midgut, with small intestine, cæcum, and ascending colon herniated into the chest, may occur in association with an extensive defect of the diaphragm, just as it may with a small posterolateral defect. This was the condition found at post-mortem examination of the infant whose diaphragm is illustrated (*Fig.* 2), and at the operations by Neville and Clowes. Beckman (1909) and Hackwood (1934) have each reported a case of a young adult with an extensive defect of the left hemidiaphragm complicated by enormous gastric distension and vomiting; and both cases were found during exploratory operations to have the small intestine, the cæcum, and the proximal colon in the chest. Hackwood's patient died a few days after

operation from continued vomiting. It would seem probable that gastric distension and vomiting were caused in these cases, as they may be in cases of herniation through posterolateral defects, by the duodenum becoming obstructed by a peritoneal band crossing it from the colon.

Campbell (1953) found one of these large defects in an elderly man during an operation for intestinal obstruction from a cause quite unrelated to the diaphragmatic defect, which had never been the cause of any symptoms. In this case the cæcum was in the normal position and the midgut normally attached. It is possible that a very large defect, if not rapidly fatal in infancy and if not associated with non-fixation of the midgut, is more likely to be compatible with a long trouble-free life than is a small defect. With a large defect there should be no risk of intestines becoming obstructed as they pass through the defect, and herniated viscera, being able to move more freely from chest back to abdomen than they are with a small defect, should allow better lung expansion and lung function.

SUMMARY

There are three types of congenital diaphragmatic defects: (1) Posterolateral defects. (2) Retrosternal defects. (3) Extensive defects involving most of a hemidiaphragm.

Posterolateral defects are situated at the periphery of the diaphragm in the region of its attachment to the tenth and eleventh ribs. There is usually free communication between the pleural and peritoneal cavities. Only rarely is there a pleuroperitoneal hernial sac. In the majority of cases urgent operative treatment is required as soon as possible after birth. The condition can be diagnosed by having chest radiographs taken of newborn babies with dyspnœa, for the presence of gut in the chest can be recognized on a plain film. In those who survive beyond early infancy, dyspnœa is again the commonest symptom, but there may also be vomiting. A thoraco-abdominal incision in the line of the tenth interspace is recommended as the best operative approach. There are two points of particular importance in operating on these cases. First, the midgut is usually not attached to the posterior abdominal wall and may be malrotated, and in these cases it is advisable to carry out Ladd's procedure (division of the peritoneal fold passing across the duodenum from the hepatic flexure or the malrotated cæcum) in order to prevent the later development of duodenal obstruction or midgut volvulus. Secondly, the use of excessive pressure in attempts to inflate the lung should be avoided, for this may rupture alveoli and cause a tension pneumothorax. There may be some difficulty, after reducing the herniated viscera and suturing the defect in the diaphragm, in closing the abdomen, which, as it has been without its usual contents, is smaller than normal; but in all the cases described a one-stage closure proved possible. An important point in post-operative care, especially of infant cases, is the withholding of oral feeds until the tense abdomen becomes soft.

Retrosternal hernias do not, as a rule, give rise to symptoms; but they may cause obstruction in later life. In the cases described a midline epigastric incision was employed and simple suture of the defect carried out.

Only one example was seen of an extensive defect of a hemidiaphragm. This was an autopsy specimen from an infant who died shortly after birth.

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CONGENITAL POSTEROLATERAL DIAPHRAGMATIC HERNIA IN THE ADULT

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CONGENITAL posterolateral diaphragmatic hernia in adults is rare, and in this differs from the well-recognized condition in infancy. In adults the commonest site is at the œsophageal hiatus, and although probably congenital in origin only presents at a much later date. In infancy, while a posterolateral defect is quite common, an œsophageal hernia is relatively rare and herniæ through the foramen of Morgagni are uncommon in either age-group.

Many series of cases of congenital diaphragmatic hernia in infancy have been reported in the literature (Harrington, 1948, 372 cases; Roe and Stephens, 1956, 47 cases; Moore, Battersby, Roggenkamp, and Campbell, 1957, 16 cases; and Koop and Johnson, 1952, 15 cases). On the other hand, the total number of reported adult cases is merely 34, the biggest personal series being that of Boyd and Wooldridge (1955) who saw 4 cases. The largest number of collected cases to be discussed in any one publication is 9 (Liebow and Miller, 1940) and therefore, because there is so curiously little cross reference by the authors interested in the subject, the various publications are here gathered and compared. The case to be described exhibits with tragic clarity the common failure of diagnosticians to keep this condition in mind; it shows the possible significance for life or death of an old case report that had been preserved for 45 years; and it recalls that this congenital abnormality, so lethal in infancy, may sometimes be silent until later life.

Embryology.—The earlier views on the formation of the diaphragm were reasonably well incorporated in Dunhill's study (1935); variants of his view were later proposed by Liebow and Miller (1940), Rives and Baker (1942), and Bremer (1943). More recently the whole subject has been again studied and the results of this intensive and extensive work by Wells (1954) are significantly related to the problems of diaphragmatic hernia.

Diagnosis.—Diagnosis in the infant is of particular urgency, and as Liebow and Miller (1940) and Moore and others (1957) point out, the signs are essentially cardiorespiratory, typically as the triad: dyspnœa, cyanosis, and 'dextrocardia'. In the adult such symptoms as occur seem to depend mainly on obstruction of the stomach or intestine; these can be extremely misleading (*Table I*), not least because of their slow and insidious development (Barrett and Wheaton, 1934). Other symptoms in the adult are pain in the side of the chest, myocardial infarction-like pain, dyspnœa, hæmatemesis and melæna leading to a chronic anæmia, chronic cough, and palpitations.

Table I.—MISLEADING SYMPTOMS IN CONGENITAL DIAPHRAGMATIC HERNIA IN ORDER OF FREQUENCY (*After Harrington*, 1942)

Abdominal	*Intrathoracic*
Cholecystitis	Cardiac disease
Cholelithiasis	Emphysema
Gastric ulcer	Congenital cystic lung
Duodenal ulcer	Pleural effusion
Secondary anæmia	Tuberculosis
Carcinoma cardia	
Stricture of œsophagus	
Appendicitis	
Intestinal obstruction	

Radiological investigations themselves can be misleading and the straight radiograph has been interpreted as cystic lung or resolving pneumonia, while the usual absence of adhesions and consequent easy reduction of the hernia has meant that the barium shadows are not likely to be in the thorax unless the patient is put in the Trendelenburg position (Dunhill, 1935).

The reported cases emphasize the value of the old saying that in every chronic case the diagnosis should be periodically and completely revised.

Treatment.—In adults immediate operation is strongly advised. Opinion is divided on the correct surgical approach, although most authors (Gross and Ladd, 1953; Harrington, 1948) use the abdominal approach for left herniæ, and for right herniæ the thoracic approach sometimes combined with a phrenic crush. Boyd and Wooldridge (1955), however, used a thoracic approach on both types in their 4 cases. Pre-operative treatment and observation as advised by Meyer (1950) is an integral part of any operation.

CASE REPORT

The present patient, Mrs. J. D., died in January, 1958, having survived for 64 years with a massive left posterolateral congenital diaphragmatic hernia.

HISTORY.—In 1912, then aged 19 years, she was admitted to this hospital with a 2-month history of recurrent abdominal pain and vomiting after meals. The pain began in the abdomen and radiated to the left side. Before this she had no trouble apart from "rumbling in the left side which comes and goes but causes no inconvenience".

On examination no abnormality was detected in heart or lungs; there was very little movement of the abdomen on respiration. The stomach was palpable at the level of the umbilicus and very marked rumbling was heard. Chemical analysis of gastric juice showed free acid and the test for fæcal occult blood was negative.

A radiograph on Oct. 4, 1912, showed the "stomach perfectly atonic, greater curvature reaches pubis".

On Oct. 8, 1912, laparotomy was performed by the late Mr. A. Don under chloroform and ether. "Abdomen opened through left rectus incision. Stomach very much dilated with distended vessels on its surface: it nearly filled whole of abdominal cavity and pelvis: no sign of an ulcer. The small intestines were absent: the duodenum was present but enlarged and passed into a narrow descending colon. No appendix. No cæcum. No omentum.

"Liver was slightly prolapsed. Pancreas present but small. Spleen felt normal and also kidneys. Genital organs present and normal.

"After these findings the abdomen was closed in normal way. Dry dressings.

"Post-operative condition satisfactory.

"Conclusions: Stomach quite atonic. Greater curvature reaches pubis. Peristalsis very slight.

"Disease: Diaphragmatic hernia."

[The operation note and conclusions are given verbatim because of the last surprising statement. Nowhere else in the case sheet is diaphragmatic hernia mentioned nor is the site of a hernia described at operation—in fact one might conclude that a congenital absence of the small intestine had been suspected—yet at the very end of the case sheet the correct diagnosis is baldly stated.]

The patient was discharged 11 days after operation and no further record can be found until 1940, when at the age of 47 years, having married and produced two daughters and a son, she presented with a history of pain in the left chest, shoulders, and back for two weeks, swelling in the front of her neck, and a choking sensation for nearly six months. The swelling in her neck had decreased in size after treatment with iodine and she had felt quite well until two weeks before admission on Nov. 13, 1940.

Previous history included pleurisy as a young girl and an operation for gastric ulcer when aged 19 years.

On Examination.—No significant cardiovascular abnormality. Blood-pressure was 130/90. Chest expansion was moderate and the percussion note was diminished over the left apex and over the middle of the left lung. Breath-sounds and vocal fremitus were both diminished in these areas. Abdominal examination was negative. The results of biochemical and hæmatological examinations were normal.

Fig. 17.—Radiograph reported as showing massive effusion on left side of chest displacing heart to the right.

Four separate X-ray examinations were made and showed basal consolidation, at first thought to be tubercle, later thought to be fluid; this gradually resolved over a period of four weeks and the patient was thereafter discharged on Dec. 7, 1940 with a diagnosis of unresolved pneumonia.

In July, 1954, she reported as an out-patient with a history of persistent hoarseness after an attack of influenza. She had later developed a choking sensation with a feeling of a lump in her throat, especially when lying on her left side in bed. A diagnosis of a large colloid goitre was made, with possible tracheal displacement and intrathoracic extension. A radiograph of the thoracic inlet showed a pretracheal opacity causing anteroposterior and probably lateral compression of the trachea, and a large left pleural effusion without mediastinal displacement.

As the patient had clinically and radiologically the signs of a left pleural effusion she was admitted, and a chest tap was attempted unsuccessfully. As will be seen from the next report, this confused rather than helped diagnosis.

Radiography showed that "penetrating films confirm the presence of a little effusion on the left side and raise the question of underlying cystic changes in the lung. These areas, however, may be due to air allowed in during the needling of the chest. No evidence of intrathoracic goitre."

During the next year the patient began to lose weight, despite a good appetite, and had increasing breathlessness and tiredness. She continued as an out-patient on conservative treatment until March, 1957, when operation was advised as the enlarged thyroid was thought to be causing pressure symptoms.

Fig. 18.—Post-mortem examination, showing left pleural cavity filled with grossly distended small and large bowel, and abdominal cavity filled by grossly distended stomach.

Prior to her admission a further radiograph was taken (*Fig.* 17). "A fairly massive effusion is present on the left side of the chest displacing the heart over to the right. Cystic changes are present inside this pleural effusion and it is noted that in 1954 the presence of cystic change of the lung was queried."

On admission, the thyroid was found to be diffusely enlarged. There were no eye signs and no tremor of the hands; the palms were warm and moist. Pulse-rate was 88–92 per minute.

Examination of the cardiovascular system showed no abnormality apart from an impalpable apex and a blood-pressure of 190/100. Respiratory examination revealed that the trachea was deviated to the left and that respiratory movements, percussion note, breath-sounds, and vocal fremitus were diminished on the left side.

On March 8, 1957, the patient had an uncomplicated partial thyroidectomy and was discharged 14 days later. Pathological examination showed the appearances associated with thyrotoxicosis.

Three months later she reported once more as an out-patient with a 4-week history of increasing breathlessness and swelling of the face, particularly around the eyes. She was re-admitted for further investigation and treatment. More detailed questioning revealed that she had had increasing flatulence and occasional tightness in the

Table II.—PUBLISHED CASES OF ADULT CONGENITAL DIAPHRAGMATIC HERNIA

No.	Reference	Sex and Age	Symptoms	Course
1	Bowditch, 1853	M. 17	Occasional 'stitch in side'. Attacks of unconsciousness	Patient struck by derrick with which he was working
2	Thoma, 1882	M. 50	No gastro-intestinal symptoms	Died of pneumonia of right side. Left lung compressed by herniated viscera
3	Abel, 1894	F. 33	Had borne 7 children. Pains in left side for several years	Immediately after over-indulging in food, patient seized with severe abdominal pain and vomiting. Died 2½ mth. after operation. Volvulus of œsophagus
4	Vogel, 1913	M. 47	Abdominal pain and symptoms referable to stenosis of large bowel. Peritonitis. Died	Died of peritonitis due to diverticulitis of the sigmoid colon. 15 cm. left posterolateral defect. Collapsed left lung. Hernial contents: transverse colon and cæcum
5	Monks, 1914	M. 43	Died about 1 yr. after head injury. Post-mortem to exclude injury as cause of death	Post-mortem. Left posterolateral hernia. Contents: stomach, transverse colon, left lobe liver. Cause of death: pneumonia
6	Kirmisson, 1914	M. 12	Pain in left chest. Palpitations. Vomiting. X-ray left diaphragmatic hernia	Operation: hernia repaired. Contents: small intestine, cæcum, and inflamed appendix which was causing symptoms
7	Borden, 1922	F. 25	Pertussis age 7 yr. Long history of recurrent abdominal pain. Acute attack suggesting appendicitis. Indigestion and shortness of breath	1917, tonsillectomy 1918, left pleurisy 1920, operation, reduction of large hernia and suture of diaphragm. Cured
8	Unger and Speiser, 1926 *Case* 1	M. 36	Seized suddenly with sharp abdominal pain and hæmatemesis. Died on admission. No previous history	Post-mortem. Hernial orifice 7½ in. diameter. No sac. Hernia consists of strangulated small bowel, transverse and descending colon. Stomach dilated. Compression atelectasis of left lung. Hæmorrhage into lumen of intestine
9	*Case* 2	M. 43	Working as blacksmith. Seized with vertigo and abdominal pain, and pain in left lower chest. Previous history of increasing cough and dyspnœa for 7 yr. Died	Post-mortem. Left lung compressed by dilated strangulated stomach. Hernial orifice 3½ in. diameter. No hernial sac. Hernia also consisted of splenic flexure and duodenum
10	*Case* 3	F. 48	No symptoms. No serious illnesses. 4 pregnancies, 1 stillbirth; 3 children alive and well; no complications recorded. Fracture of humerus in accident	Massive hernia discovered on X-ray examination. Left lung collapsed except for small area at apex. Compensatory emphysema right lung. Calcified glands left upper chest. Hernia probably consisted of colon and stomach. Refused further investigation and treatment. Recovered
11	Lilienthal, 1931	F. ?	16 yr. previously diagnosed 'tumour of the chest'. Bronchoscopy negative. Operation 6 yr. before for ectopic. Has 5-yr.-old child. Complaint: heaviness in right chest, slight difficulty with breathing. Dry cough. Diagnosis: mediastinal dermoid	Operation. Right thoracotomy. Globular mass in posterior diaphragm in costophrenic junction. Deformed right lobe liver covered by peritoneum. Phrenic crush. Hernia closed. 9 mth later well, but X-ray showed some 'tenting' of diaphragm
12	Geyman, 1931	F. 72	Cholecystectomy at age 52. 9 normal pregnancies. Complaint: obesity, constipation, rheumatism. Examination: dull right lower chest; absent breath sounds. X-ray: mass right lower chest; barium showed hepatic flexure and transverse colon in thorax	No record of course or treatment. No confirmation of diagnosis
13	Dunhill, 1935 *Case* 1	F. 52	Retrosternal pain and dysphagia for 2–3 mth.	Operation: hernia left posterior quarter of diaphragm. No hernial sac. Cured
14	*Case* 2	Adult	Full details not given. All had severe abdominal pain. One also had severe pain in left shoulder. One had a hæmatemesis	All operated on. Pleuro-peritoneal hernia. Hernial sac present. All cured symptomatically. One case also had gall-stones. Had previous cholecystectomy when hernia was missed as it was then in reduced state
15	*Case* 3	Adult		
16	*Case* 4	Adult		
17	Forty, 1935	M. 70	Abdominal pain and vomiting for 6 d. Previous health good	Operation: hernia in right dome of diaphragm containing terminal 3 feet of ileum. Died post-operatively. Post-mortem. Opening 2½ in. diameter. No sac. No adhesions
18	Harrington, 1942 *Case* 1	Adult	No details given	Hernia through pleuroperitoneal canal—relieved
19	*Case* 2	Adult	No details given	Hernia through pleuroperitoneal canal—relieved
20	*Case* 3	Adult	No details given	Hernia through posterior quarter left diaphragm. Died
21	Rives and Baker, 1942	F. 19	Abdominal pain, nausea, and vomiting. 'Smothering spells' until 4½ yr. old	Right diaphragmatic hernia. Pleuroperitoneal membrane intact. First operation, 1936. Recurrence 1½ mth later. Second operation, 1937. Relieved. X-ray satisfactory 3 yr. later

Table II.—PUBLISHED CASES OF ADULT CONGENITAL DIAPHRAGMATIC HERNIA (*continued*)

No.	Reference	Sex and Age	Symptoms	Course
22	Shearer, 1945	M. Adult	New Zealand soldier. February, 1942: X-ray diagnosis, hydatid cyst. March, 1943, well. Chest N.A.D. clinically. 1944, occasional pain right side chest on exertion. July, 1944, mass still present above diaphragm	June, 1942, thoracotomy. Tumour found to be liver. No record of further treatment
23	Bonniot and Flandrin, 1953	M. 52	"Gros buveur'. Served in Army 1939–40. 1949, pain in left hypochondrium after meals. Some dyspnœa. Hernia diagnosed. 1952, severe dyspnœa. Signs of obstruction	Operation, Sept. 1952: congenital defect in left diaphragm, repaired with nylon sutures. Hernia cured. Developed severe anæmia
24	Joussemet, 1953	M. 39	Presented 1949 with atypical digestive symptoms. Hernia diagnosed. Operation refused 1952. Signs of obstruction	Operation. Hernia through pleuroperitoneal canal. Gross distended intestines. No hernial sac. Died during operation, ? ileus
25	Mars and Bollinger, 1953	F. 67	Recurrent backache 15 yr. Symptoms continued. Backache continued. Presented in 1953 with 2 d. history epigastric pain, coffee ground vomiting, and melæna. X-ray: mass right thorax. Provisional diagnosis: œsophageal hernia	Operation, 1940: spinal fusion. Operation, 1943: cholecystectomy—no relief. Operation, 1953: left oblique subcostal incision. Right posterior defect in diaphragm 2 × 3 × 4 cm. Stomach herniated. No hernial sac. Cured backache
26	Ransdell and Ellison, 1953	F. 21	Mild lifelong dyspnœa increasing over past 2–3 yr. Admitted with pain in chest and stomach. Nausea, vomiting. Hacking cough. X-ray: right diaphragmatic hernia; barium enema of colon and small intestine	Emergency right thoracotomy. Defect posterolateral 5 cm. diameter. No sac. Strangulated bowel covered with fibrin. Volvulus middle lobe right lung. Middle lobe resected. Hernia closed with difficulty. Died post-operatively
27	Boyd and Wooldridge, 1955 *Case* 1	M. 54	Asymptomatic. Tumour in right chest found during X-ray survey. Physical examination non-contributory. Pre-operative diagnosis: neurofibroma	Operation: right thoracotomy. Tumour of retroperitoneal fat 5 × 7 × 7 cm. Posterolateral defect 5 cm. in diameter. No peritoneal sac. Closed. Recovered
28	*Case* 2	M. 57	Right upper abdominal pain for 6–7 wk. X-ray of gall-bladder negative. Egg-size mass in right posterior mediastinum. Pre-operative diagnosis: neurofibroma or hernia	Operation: right thoracotomy. Posterolateral defect. Tumour. No peritoneal sac. Hernia small and easily closed. Recovered
29	*Case* 3	F. 13	Exertional dyspnœa since age of 3 yr. Occasional cyanosis and pain in left chest. Acute attack following colonic irrigation. X-ray small intestine and large bowel in left chest	Operation: left thoracotomy. Defective posterolateral 6 in. in diameter. Small intestine and large bowel in thorax. No hernial sac. Atelectasis left lower lobe. Closure difficult. Recovered
30	*Case* 4	M. 65	Chronic cough for 10 yr., increasing for 12 mth. Some exertional dyspnœa. Bowel sounds audible left chest. X-ray: bowel gas and increase of density. Bronchoscopy, bronchogram. Collapsed left lower lobe	Operation: left thoracotomy. Left lower lobe collapsed. Hernia of transverse colon through left posterolateral defect. No hernial sac. Defect large. Closed with fascia lata. Recovered. Cough ceased
31	Roe and Stephens, 1956 *Case* 1	F. 30	9 mth. pregnant, sent to psychiatric clinic because abdomen flat. Delivered full-time soon afterwards. Hernia diagnosed in puerperium. Asymptomatic	Operation in puerperium. Abdomen contained only uterus. Contents replaced. Hernia repaired
32	*Case* 2	F. 35	Housewife. Only complaint recurring hæmoptysis. X-ray ill-defined, density left lower chest. Bronchoscopy negative. Barium enema: large hernia	Operation. Pleural cavity contained large and small bowel including cæcum and appendix. No hernial sac. Left lower lobe lung hypoplastic. Contents reduced. Hernia closed. Post-operatively: well 5 yr. later. No hæmoptysis
33	St. Raymond, Hardy, and Robbins, 1956	M. 13	Recurring abdominal pain for 1 yr. Acute attack with nausea and vomiting for 9 d. X-ray chest: translucent area of left lower chest	Operation: left thoracotomy. Posterior diaphragmatic hernia containing stomach and spleen. Hernial sac present. Accessory lobe of left lung communicating with œsophagus. Lobe resected. Hernia repaired. Recovered
34	Rakower, Milwidksy, and Wayl, 1956	M. 12	1951, mass radiography. Lower two-thirds left chest opaque. No previous history. 1952, diagnosis, pleural effusion. Chest tap attempted. Patient fainted. Barium meal and enema: posterior hernia small intestine, cæcum, colon. Bronchoscopy: hypoplasia left lung	Operation. Left pleural cavity almost filled with intestine. Posterior defect 10 × 6 cm. No sac. Many adhesions at hernial margin. Left lung hypoplastic. Recovered. Well 2½ yr. post-operatively. X-ray lung: good expansion

chest and tingling in the fingers of the left hand. Appetite was good, weight increasing, and there was no dysphagia.

The physical findings on this admission were practically unchanged, there still being signs of an effusion of the left base. Abdominal examination revealed no abnormality. There was no hernia. A further chest tap was performed without success. Treatment with thyroid improved her condition and she was discharged three weeks later feeling much better, on a maintenance dose of thyroid gr. ½ t.d.s.

On Jan. 25, 1958, she was admitted for the last time, as an emergency, with a history of increasing breathlessness for one week. She was extremely cyanosed and collapsed and died a few minutes after admission before any treatment could be instituted.

POST-MORTEM.—Examination of the head and neck was negative, apart from marked scarring at the site of the thyroidectomy, and no obvious thyroid tissue was found.

The left pleural cavity was filled with grossly distended small and large bowel. The herniated bowel consisted of all the small intestine from the duodenojejunal flexure, the cæcum, appendix, ascending and transverse colon to the splenic flexure where the bowel was kinked and causing an obstruction. The large bowel distal to the splenic flexure was collapsed and practically devoid of fæces (*Fig.* 18).

In the posterolateral aspect of the left diaphragm there was a large hernial opening 7 cm. in diameter. The margins of the hernial orifice were perfectly smooth, and posteriorly a small ridge of muscle could be felt on the costal margin. There was no hernial sac.

The abdominal cavity was practically filled by the grossly distended stomach which extended from the left hypochondrium to the right iliac fossa. The duodenum, which was also distended, did not pursue its normal C-shaped course, but travelled in a superolateral direction to the medial margin of the hernial orifice.

Complete reduction of the herniated bowel was impossible as the mesentery was attached to the anterior surface of the left kidney, the upper half of which was contained within the pleural cavity. After some dissection, reduction was achieved and revealed a smooth pleural cavity containing an extremely small and collapsed left lung (weight 88 g.).

The heart (335 g.) and mediastinum were slightly displaced towards the right, the cardiac apex being 3 cm. to the left of the midsternum. The right lung (550 g.) was grossly congested and emphysematous with numerous bullæ on its pleural surface.

The spleen (90 g.) was at the anterior margin of the hernial orifice, but was contained within the peritoneal cavity, as was the normal liver (1425 g.). The upper half of the left kidney (155 g.) was within the pleural cavity, and on section the whole organ was grossly congested. The right kidney (265 g.), although rather pale by comparison, appeared normal apart from a large cyst 5 cm. in diameter in its lower pole.

The remaining organs were normal both in situation and appearance and the only other abnormality was some atheroma of the lower third of the aorta.

HISTOLOGY.—The main organ of interest is the left lung which shows areas of collapse alternating with areas of emphysema. The lung structure is normal and judging from the amount of carbon pigment present it must have functioned quite satisfactorily during life. The only other change seen is considerable congestion with many bronchi containing amorphous debris, polymorphonuclear leucocytes, and numerous colonies of bacteria.

The right lung shows many areas of emphysema and collapse with a large amount of œdema fluid and red cells in the alveoli which have lost their nuclear staining. The bronchi are stuffed with amorphous debris, some of which is probably bile, and the appearances suggest inhalation of gastric contents with massive collapse and necrosis of adjacent alveoli.

A section from the site of the previous thyroidectomy shows severely atrophied thyroid surrounded by a mass of fibrous tissue and heavily infiltrated with lymphocytes and plasma cells. There is no obvious functioning thyroid present and it is not surprising that the patient was myxœdematous in the last few months of her life.

The remaining organs are histologically normal.

CASE SUMMARIES

Having found no previous review of the subject of adult congenital diaphragmatic hernia, I have endeavoured to list all the published cases (*Table II*). The smallness of the total is due in part to the inclusion only of those cases clearly designated as 'adult congenital'—a distinction that has not always been made.

Analysis of Case Histories.—

Symptoms.—The symptoms cover a wide range (*Table III*) and vary greatly in frequency from abdominal pain (17 cases) to backache (1 case). The low incidence of severe or moderate respiratory symptoms (7 cases) is noteworthy and in sharp contrast with the usual picture in the infant. Although Roe and Stephens (1956) make the point that respiratory function can in some cases increase to satisfactory levels, the significant fact for the clinician is the number of cases which have virtually no evidence of any upset of respiratory function. Indeed, it seems likely that this is one of the main reasons why the condition is so often absent from the differential diagnosis.

Table III.—SYMPTOMATOLOGY IN 34 RECORDED CASES

	No. of Cases
None	7
Abdominal pain	17
Vomiting	9
Obstruction	3
Dysphagia	2
Dyspnœa	7
Other cardiovascular symptoms	8
Chest pain	8
Backache	1

Table IV.—CASES WITH LONG CLINICAL HISTORY

Case No.	*Age in Years*	*Duration of Symptoms*
1	17	Probably since birth
7	25	Long history of recurrent abdominal pain
21	19	Smothering spells since birth until 4½ years old
26	21	Dyspnœa increasing in past 2–3 years
29	13	Dyspnœa since age of 3 years
5 cases	Average age 19 years	

Duration of Symptoms.—Comparison of the cases giving a long clinical history (*Table IV*) with those having no clinical history or a long symptomless history (*Tables V* and *VI*) reveals a sharp difference in age in the two groups. In the former, the ages vary from 13 to 25 years, average age 19 years, whereas in the previously symptomless group, the ages vary from

Table V.—NO CLINICAL HISTORY

Case No.	*Age in Years*	*How Diagnosed*
2	50	Post-mortem
5	43	Post-mortem
10	48	Fracture of humerus; on examination
27	54	X-ray survey
31	30	Following pregnancy
34	12	X-ray survey
6 cases	Average age 39 years	

Table VI.—Cases with Long, Previously Symptomless, History

Case No.	*Age in Years*	*Symptom-free (years)*	*Duration of Symptoms*
3	33	25	8 years
4	47	47	A few days
6	12	12	Hours
8	36	36	Several hours
9	43	36	Dyspnœa for 7 years. Abdominal pain several hours
12	72	?72	Not given. Cholecystectomy at 52 years
13	52	52	2–3 months
17	70	70	6 days
23	52	49	Intermittent for 3 years
24	39	36	Intermittent for 3 years
25	67	52	15 years
28	57	57	6–7 weeks
30	65	55	10 years
33	13	12	1 year
14 cases		Average age 47 years	

12 to 72 years, average age 44 years, these latter patients frequently presenting with acute symptoms.

Treatment.—Despite the seriousness of the condition and the hazards of a major operation in the past, the results of treatment are remarkably good—20 patients relieved and 5 deaths. With the increased frequency of operation for the infantile type of case and the consequent improved understanding of the local mechanics, it is probable that even better results will be obtained in adult cases in the future.

DISCUSSION

Although adult congenital hernia is a rare condition, enough cases have now been reported to reveal its nature and its importance in the differential diagnosis of obscure abdominal or intrathoracic symptoms.

The hernia occurs in either side of the diaphragm, but more commonly on the left side, probably because of the so-called protective action of the liver on the right. A sac is usually absent and the contents of the hernia are thus in direct contact with the thoracic viscera.

The facts now available suggest that congenital diaphragmatic hernia should be regarded as occurring in two forms. These are somewhat similar to multiple cystic disease of the kidney in that one form manifests itself at the beginning of life and the other possibly much later. Both forms of congenital diaphragmatic hernia are eminently treatable, but although the fundamental abnormality is probably identical the symptomatic picture is profoundly altered by the age of the patient. The sudden intense distress, often respiratory, that characterizes the clinical picture in the infant is very different from the grumbling remittent and misleading symptoms of the adult. Moore and others (1957), for instance, reported 16 cases of congenital hernia in infants whose ages ranged from 45 minutes to 90 hours, all of whom presented with dyspnœa and cyanosis.

Even if the clinician has thought of the possibility, he may be misled if the radiologist is not also primed, for a barium investigation can fail to reveal the hernia if the patient is not in the Trendelenburg position. Indeed, even at laparotomy the surgeon can be deceived, since the viscera concerned may have slipped back into their normal site. This free movement of the viscera between the pleural and the peritoneal cavities certainly plays a large part in the protean character of the symptoms, but this very variety may in fact be most helpful to the clinician.

In the present case it is probable that some reduction of the hernia occurred periodically and, combined with the powers of adaptation of the respiratory and cardiovascular systems, thus allowed the patient to lead a normal existence. It is worthy of note that the abdominal distension occurring in pregnancy did not aggravate the symptoms in the present case or in the two other reported cases (*Cases* 10 and 31). Acute or subacute obstruction of the herniated bowel no doubt could explain some of the respiratory or cardiovascular symptoms that occasionally add to the misleading clinical picture of this curious condition.

The case history presented is an argument for the preservation of clinical records, and even more so for their ready availability and perusal.

SUMMARY

A case of adult congenital posterolateral diaphragmatic hernia is reported, and 34 cases from the literature examined.

A brief review is given of the symptoms, signs, radiological diagnosis, and appropriate treatment.

Analysis of the reviewed cases suggests that patients who survive infancy may have only vague or insignificant symptoms until later life, when an abdominal or intrathoracic disorder is only too often unrecognized as this dangerous but treatable condition.

Addendum.—Since this article was prepared a paper has been published in the *British Journal of Clinical Practice* (McDougall, C., 1958, **12**, 878) which describes the case of a man of 65 years whose only previous history was of bronchitis and attacks of asthma since his late teens and who died during an attack of status asthmaticus.

Reference is given also to 3 further cases (Le Wald, 1927; Hackwood, 1934; Barclay, 1936).

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HERNIATION THROUGH THE FORAMEN OF MORGAGNI

BY W. R. HUNTER, LIVERPOOL

HERNIÆ of all types still present many problems of diagnosis and treatment, but none more so than the internal varieties. These difficulties apply especially to herniation through those openings in the diaphragm known as the foramina of Morgagni, since the aetiological factors concerned remain obscure and the symptoms presented are so bizarre. From the literature it appears that these foramina are rare sites for herniation to occur; indeed Saltzstein, Linkner, and Scheinberg (1951), when reporting their case, could collect only 44 others; and Harrington (1951), in his own series of 534 patients suffering from all kinds of diaphragmatic herniæ, on whom he had operated, found only 14 of this type, 2 of which were bilateral.

This form of hernia, however, is not so uncommon as the literature suggests, and not infrequently cases are found when a systematic search for the condition is carried out; 2 of the 5 cases here recorded were revealed by chance when investigations were being undertaken for other diseases. There is little doubt that with the increase in the routine use of chest radiographs, further instances of this unusual hernia will be accidentally discovered, as is already apparent from the report by Cruickshank, Goel, and Macedo (1954), who observed 6 cases from their chest clinics.

The hernia may cause minimal or no complaints, and so occasion little inconvenience to the patient; on the other hand the symptoms may be severe and acute, and as the treatment is not only easy but a life-saving procedure, recognition of the condition is of great importance.

CASE REPORTS

Case 1.—Miss A. M., born 1871, was first seen on Nov. 20, 1952. She complained that for the past nine months she had had occasional attacks of abdominal discomfort. There had never been any vomiting, her bowels had been regular, and she had no other symptoms.

In the last three weeks she had vomited frequently and had had upper abdominal pain. These symptoms had become increasingly worse and she was now unable to keep any food down. The vomiting started after she had slipped off a ladder.

ON EXAMINATION.—She was a fairly well-nourished but dehydrated old lady. There was no abdominal distension and no external herniæ, but some tenderness on deep pressure in the right upper abdomen. Although she was vomiting it was decided to do a barium swallow, and the following was the report: "The cardiac end of the stomach is normally placed below the left side of the diaphragm. The œsophagus enters the stomach normally. There is a volvulus of the stomach so that the pyloric end is situated on the right side at a higher level than the fundus. The right side of the diaphragm is not recognizable, but I think this stomach volvulus is through a diaphragmatic hernia. There is obstruction to the outflow of the stomach, probably due to compression of the first part of the duodenum by the diaphragmatic hiatus. Very little of the meal had left the stomach up to half an hour. I did not keep the patient longer." (*Fig.* 19.)

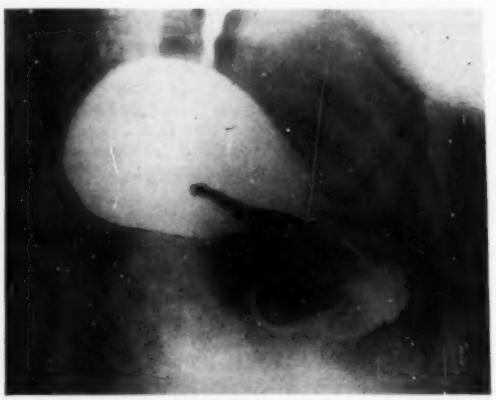

FIG. 19.—This shows a volvulus of the stomach; the pyloric end is situated on the right side at a higher level than the fundus. The œsophagus enters the stomach normally. The radiograph shows also the narrowing in the body of the stomach and the pylorus at the opening in the diaphragm.

AT OPERATION.—After intravenous saline preparation to rectify the electrolyte imbalance and dehydration, a laparotomy was done, through a midline supra-umbilical incision.

There was an oval-shaped opening, 2 in. across, in the diaphragm just to the right of the round ligament, which had all the appearances of a foramen of Morgagni.

Although stomach, omentum, and some transverse colon had entered the sac, the hernia was reduced very easily

and the contents returned into the abdomen. The stomach and colon appeared normal. By using only gentle traction the sac was delivered without difficulty from the thorax, and was excised. The oval opening in the diaphragm was closed by suturing its posterior margin to the anterior edge of the foramen, with interrupted nylon.

The patient made an uninterrupted recovery and is still alive and well six years later. She is symptom-free.

Case 2.—Mrs. M. A. B., born 1889, was first seen on March 31, 1951, and was admitted with acute abdominal pain and vomiting—she had had these symptoms for five days. Prior to this attack she had had no serious illnesses until three months ago, when she had an attack of upper abdominal pain and vomiting. This soon got better, but in the past three months she had had four similar bouts. In each instance she vomited many pints of brownish

FIG. 20.—Transverse colon seen in an anteroposterior view; it is entering the chest through an opening in the right side of the diaphragm.

FIG. 21.—Lateral view showing the colon entering the thorax through an opening in the anterior part of the diaphragm.

fluid and every attack lasted several hours. The present attack has continued for five days, with continuous vomiting and severe upper abdominal pain.

ON EXAMINATION.—She appeared shocked. The tongue was dry. There was tenderness in the right upper abdomen. It was at first thought that she was a case of pyloric obstruction, and she was treated by stomach aspiration and with intravenous fluids and electrolytes. After her general condition had improved a barium swallow was done and this showed what appeared to be a volvulus of the stomach.

AT OPERATION.—Laparotomy was performed (by Mr. Gibbon) through a right upper paramedian incision. A diaphragmatic hernia was found through the foramen of Morgagni on the right side. The neck of the sac was about 2 in. across and the sac itself was enormous, containing half the stomach and most of the transverse colon and great omentum. These organs were easily delivered into the abdominal cavity and were found to be normal; no other abnormality was detected. The margin of the sac was incised and the sac was then easily withdrawn from the thorax. The upper edge of the opening in the diaphragm was sutured to the costal margin with nylon, so covering and obliterating the opening.

The patient made a satisfactory recovery and six years later is well and symptom-free.

Case 3.—Miss S. E. G., born 1892, was first seen by Mr. Helsby on Feb. 20, 1957. She complained of a pain under the right shoulder blade which radiated into the right upper abdomen. The pain started suddenly in the night about eight weeks previously, and had recurred at intervals since. It was not related to food or exercise, lasted up to three hours, was not related to posture, and in the first instance she had not done any heavy lifting the day previously. She had no dyspeptic symptoms, the bowels were regular, there was no cough or breathlessness. There was nothing of importance in the previous history. Her doctor suspected that there might be something wrong with the chest and had a radiograph taken. This revealed a shadow in the right lower chest which the radiologist thought might be due to bowel, and in the nature of a diaphragmatic hernia.

ON EXAMINATION.—There was some degree of anæmia, but nothing amiss in the chest or the abdomen. A barium meal was reported on as follows: "*Chest*: There is no evidence of any pulmonary pathology, but there is a large hernia through the foramen of Morgagni on the right side. The hernia contains a loop of colon and is not causing any obstructive lesion. There is a diverticulosis of the pelvic colon, otherwise the gastro-intestinal tract shows normal appearances." (*Figs.* 20, 21.)

AT OPERATION (Professor Wells).—The abdomen was explored through a midline incision. There was a sub-costosternal hernia on the right side. The opening was 2 × 1 in., and the sac contained transverse colon and omentum which were easily reduced. The cavity was about 6 in. deep. The gall-bladder and stomach were normal.

The greater part of the sac was excised. A narrow cuff of peritoneum around the foramen was everted and the peritoneal opening closed with a continuous catgut suture. The edges of the foramen were then approximated, superficial to the previous suture line, with interrupted thread sutures. This was accomplished without tension.

FIG. 22.—Transverse colon seen in an anteroposterior view; it is entering the chest through an opening in the right side of the diaphragm.

FIG. 23.—Lateral view showing the anterior opening in the diaphragm, with the transverse colon passing through it into the chest.

The patient made an uninterrupted recovery apart from a little hæmatoma in the wound. She has been free of all symptoms since the operation.

Case 4.—Miss R. S., born 1876, was first seen in 1937 complaining of upper abdominal pain, and on examination was tender in the right upper abdomen. At this time she had a Graham's X-ray examination which showed a non-functioning gall-bladder, and radiograph of her chest demonstrated slight elevation of the diaphragm on the right side and the appearances of an unresolved pneumonia.

A cholecystectomy was done, but no mention was made in the operation notes of any evidence of a hernia at this time, but as the cholecystectomy was an exceedingly difficult one the hernia was not specifically looked for.

Six months later the patient was symptom free, but the radiologist was still puzzled as to why there was what appeared to be an unresolved pneumonia on the right side. These radiographs are not now available, but the reports show there was some unexplained shadowing persisting in the right lower chest.

The patient was not seen after this until 1952. She then had an acute attack of abdominal pain with diarrhœa; it was suspected that she might have a carcinoma of the colon and a sigmoidoscopy and barium enema were done. The barium enema showed a right-sided diaphragmatic hernia containing colon, with an opening in the diaphragm corresponding to a foramen of Morgagni.

As her symptoms settled and she did not want any operation, she was discharged.

In February, 1958, six years later, she was seen for a follow-up and was very well apart from a little flatulence. A barium meal showed the colon still to be in the right chest, and the chest film a shadow at the right base. (*Figs.* 22–24.)

Case 5.—Miss M. M. N., born 1916, was first seen in 1956 complaining of a cough, loss of weight, with lassitude and night sweats. The patient was found to have bilateral

FIG. 24.—Shadowing at the right base due to colon having entered the right chest.

phthisis. There was, however, a shadow in the right lower chest which the radiologist thought might be due to bowel. A barium enema was done and this showed the ascending

colon and the proximal transverse colon lying anteriorly in a hernial sac through the foramina of Morgagni on the right side (*Figs.* 25, 26).

The diagnosis was made because of the appearances of a chest film. The patient had and still has no abdominal symptoms.

DISCUSSION

The foramina of Morgagni, when present, are situated in the anterior part of the diaphragm, on either side of, and immediately adjacent to, the falciform ligament, where the muscular fibres of the diaphragm are attached to the posterior aspect of the sternum and to the costal cartilages. Occasionally the foramen is present on both sides, and Harrington observed two such cases in his own series (1951); but usually the opening is unilateral and situated more often on the right than the left side. This is a surprising and unexplained fact, especially as one would have thought that the right lobe of the liver would have afforded a greater degree of protection on the right side than is provided on the left. In explanation of this apparently curious anomaly, Aabye (1954) suggested that the pericardial attachment to the diaphragm, being more extensive on the left side, offers in consequence a greater protection and support. In the 5 cases here described the opening was on the right side in each instance. The openings are round or slit-like, with the costal cartilages and sternum forming the anterior boundary and the diaphragm the lateral margins and posterior edge. The orifices may be small, or big enough to admit three fingers.

When herniation occurs through the foramen of Morgagni there is always a peritoneal sac, indicating that the peritoneal cavity becomes sealed from the pleural cavity before the advent of herniation. Why a deficiency should appear here is unknown, but it may be that there is mal-development of the mesodermal element which forms the musculature of the diaphragm; and as the musculature develops after the closure of the communication between the pleura and peritoneal cavities, such an ontogenetic anomaly would explain the constant presence of a peritoneal sac. The anterior part of the diaphragm which is involved in this condition is formed entirely from the septum transversum, and as far as is known there is no fusion of parts in this region (Hamilton, Boyd, and Mossman, 1947); thus a weakness due to inadequate fusion of the different entities from which the diaphragm is made cannot be held responsible. There is only one small natural opening in this region, where the superior epigastric artery enters the abdomen by going between the xiphoid and costal attachments of the diaphragm. This small space, filled with areolar tissue, is known as Larrey's space; it is covered superiorly by pleura, and below by peritoneum. This may be a weak spot, and Meyer (1950) believes that increased intra-abdominal pressure, plus loss of tone in the diaphragm, might allow herniation to begin at this point. Brown (1952), who described a case of bilateral herniation in a cadaver, was able to make a careful examination of the openings. He noted that the superior epigastric vessels on each side bore a constant relationship to the anterolateral margins of the openings. He was also able to demonstrate that the sternal attachment of the diaphragm was normal and intact.

FIG. 25.—Lateral view showing the cæcum and colon in the chest with the lower ileum entering the sac.

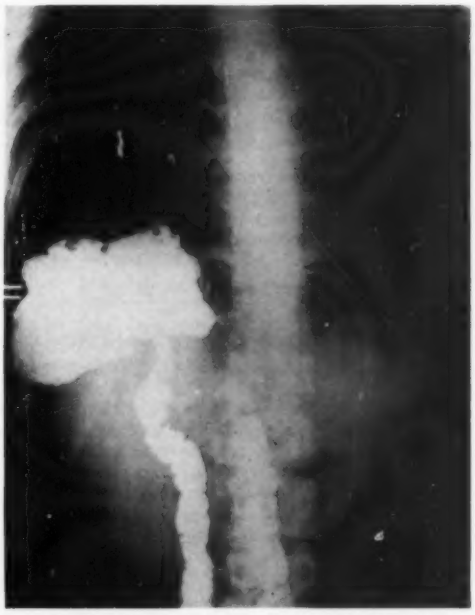

FIG. 26.—Anteroposterior view showing herniation through the right side of the diaphragm. The ileum is seen entering the sac on the right of the radiograph; the transverse colon leaving the sac on the left of the ileum.

There does appear, however, to be some relationship between this hernia and other developmental abnormalities, because in children who suffer from this condition the incidence of other congenital deformities—mongolism and feeble-mindedness—is exceedingly high.

In adults there is an appreciable number of cases in which the cæcum, ascending colon, and terminal ileum are in the sac, indicating that incomplete rotation of the gut had occurred with a high and undescended cæcum. This was commented upon by Saltzstein and others (1951), and noted in a case of Cruickshank and others (1954), and is well shown in *Figs.* 25, 26.

Colon may be present in the sac for years without giving rise to symptoms (as is illustrated in *Cases* 4 and 5), and it may be that on these occasions, in which the cæcum is in the sac, the hernia was in being before the full rotation of the gut had been completed; in this event the incomplete rotation of the gut would be secondary to the hernia. Such an explanation is conjectural, but if it were true then the association of the undescended cæcum with the hernia could be explained on grounds other than that of multiple congenital abnormalities. In the deficient state of our knowledge, however, there is as yet no satisfactory explanation of the aetiology of this hernia, nor of this intestinal abnormality commonly accompanying it, and it may be that the answer lies in a combination of congenital and acquired factors.

Although the foramina which develop at these sites are generally known as the foramina of Morgagni, various other names have been given to these openings and the herniæ through them. From time to time the hernia has been called 'substernal', 'parasternal', 'anterior diaphragmatic', and 'retrosternal', and Harrington (1945), who named it 'subcostosternal diaphragmatic', is probably the most correct in his description. The majority of the reported cases have been in adults, although the condition is found in infants and children. Saltzstein and others (1951) when they searched the literature found 44 cases; only 8 occurred in children under the age of 12 years. In these 8 there was a high incidence of other congenital abnormalities and deformities. Of the rest of the cases, 4 were in the age group 13 to 29 years, but 59 per cent were discovered after the age of 50 years. In the younger age groups males preponderated over females, but in the older age groups there was an increasing number of females, and in fact in the eldest group it was unusual to find a male.

This observation of an increasing female preponderance with increasing age is confirmed in the 5 cases here recorded, who were all women. There is no evidence that serious injury by tearing the diaphragm plays any part in the aetiology, but minor trauma may influence the symptomatology. In *Case* 1 the patient says she slipped off a ladder just prior to the onset of the acute symptoms, which might indicate that this slight injury may have caused the stomach to enter the sac, which was already formed and contained omentum and colon. Saltzstein and others (1951) in their case also noted the influence of minor trauma, and thought that the acute symptoms were due to the stomach entering the preformed sac as a result of a sudden exertion and bending forward. There is no doubt that colon and omentum may be in the sac for years and only cause the mildest of symptoms, or in fact none at all (*Cases* 4 and 5). Once stomach is involved then the symptoms are likely to be seriously aggravated and its presence in the sac usually heralds the onset of upper abdominal pain and vomiting. Stomach is rarely found in the hernia in infants and younger people and its appearance in the sac is a concomitant of advancing years, as is demonstrated in *Cases* 1 and 2, who were 81 and 62 years respectively when treated. The commonest contents of the sac are omentum and colon, and the inclusion of these organs may give rise to no symptoms or only a dyspepsia of the flatulent type, and it is interesting that seldom is large bowel sufficiently occluded to necessitate operation for intestinal obstruction.

Occasionally the patient complains of pain in the chest, as in *Case* 3, but breathlessness and dyspnœa are very unusual complaints.

If omentum is involved there are rarely any symptoms, and the solid shadow shown by the radiograph in the right chest at the cardiophrenic angle may be mistaken for a tumour in the chest.

The diagnosis is made by X rays, and the hernia may be found during routine films of the chest, or in the course of investigations on the stomach or intestinal tract. Opacities in the right lower chest at the cardiophrenic angle, and the presence of abnormal shadows in this area, especially if the patient has no chest symptoms, should make one suspicious of this variety of hernia. Cruickshank and others (1954) described 1 case in which the shadow was thought to be a thoracic tumour; when this was explored through the chest it became apparent that the repair by this approach was very difficult.

It is important when trying to demonstrate this hernia by intestinal tract radiography to take lateral films, as only by so doing will the true anterior position of the opening in the diaphragm be shown and accurately located. If the stomach is involved the barium meal reveals what at first sight appears to be a volvulus of the stomach (*Fig.* 19), and unless this investigation is done the collapse with pain and vomiting may be mistaken for pancreatitis, pyloric obstruction (Helsby and Wells, 1954), or some other upper abdominal catastrophe. If the diagnosis is in doubt pneumoperitoneum may be of great help in demonstrating whether the shadow in the thorax is in continuity with the peritoneal cavity, and this is especially useful if the hernia only contains omentum, and so cannot be shown on the barium-meal films (Clay and Hanlon, 1951).

As already mentioned, the curative operation is an easy one, but unless the stomach is in the sac or the patient is having symptoms there is no need to interfere.

An upper abdominal incision gives an excellent approach, and indeed by the transthoracic route the repair of the hernia may be awkward. In the 3 cases operated upon the contents were reduced without difficulty, and furthermore the sac was delivered into the abdomen in 2 instances with the greatest ease, so much so that the facility with which it was removed even with the slightest traction was remarkable. This fact has been noted by other writers, and especially by Aabye (1954). It is thus very important that the

condition be recognized, as the operation done through the abdomen is an easy one, and the results are good. The repair of the foramen after removing the sac is accomplished by suturing the edge of the opening together, or by stitching the posterior edge of the opening to the costal margin and the posterior aspect of the sternum, thus closing the gap. The round ligament, a most useful obturator in helping to close the deficiency, may be freed and sutured over any weakness in the repair; should difficulties be encountered the method of Parrella and Hurwitz (1949) may be adopted, whereby the opening is closed by using the transversalis fascia.

Acknowledgements.—I would like to thank Professor Wells and Mr. Helsby for their kindness in allowing me to include *Case* 3, and my colleagues at Walton Hospital for their help. Mr. Gibbon for permission to include *Case* 2, Dr. Murray for notes from the Chest Clinic about *Case* 5, and Dr. Winter for advice concerning the radiographs. Mr. Lee, of the Photographic Department, University of Liverpool, has kindly provided the photographs of the X-ray films, for which I am most grateful.

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ENDOMETRIOSIS OF THE LARGE INTESTINE

BY R. E. B. TAGART, NEWMARKET

ENDOMETRIOSIS of the large bowel is rare. Seventeen out of the 21 cases on which this report is based were found in the records of the Research Department of St. Mark's Hospital and were seen there over a period of seventeen years. Carcinomata of the rectum, on the other hand, are seen there at the rate of about 150 per year.

The remaining 4 cases in this review are from Addenbrooke's Hospital, Cambridge.

Where the incidence of particular features of the disease is concerned the totals vary, since not all the records are complete. In 5 out of 21 cases the endometrioma was discovered as an incidental lesion, the major disease being carcinoma. These cases have been excluded from the sections dealing with clinical features, treatment, and prognosis.

Three perineal lesions are included. They do not strictly fall within the scope of this report, but they were all referred to rectal surgeons and so can usefully be considered here.

INCIDENCE

J. A. Sampson (1921) believes that endometriosis of one pelvic organ or another occurs in 10–20 per cent of women between the age of 30 years and the menopause, while Masson (1945) found that the rectum or sigmoid colon was involved in 13 per cent of 2686 cases. A combination of these figures suggests that 1–2 per cent of women suffer from rectosigmoid endometriosis during their menstrual life. This high supposed incidence can only be reconciled with the clinical rarity of the condition if it is assumed that the majority of lesions never cause symptoms. In support of this assumption is the fact that in the present series nearly one-third of the lesions caused no symptoms, 5 being discovered after excision of the rectum for carcinoma and 1 in the course of an hysterectomy for uterine endometriosis.

Meigs (1949) found that pelvic endometriosis was four to five times more common among his private than his hospital patients, and he suggests that this may be connected with the greater use of contraception in the former group. However, there are other possible factors, particularly those which influence the tendency to retrograde 'tubal menstruation', namely tubal stenosis following gonococcal salpingitis, and cervical stenosis resulting from repeated intrauterine operations (Abel, 1948).

AETIOLOGY

Sampson gave the first full description of endometriosis in 1921, but the question of how functioning endometrial tissue comes to be found in sites other than the normal has not yet received a satisfactory answer.

The possibilities are: (1) Cellular metaplasia; (2) Venous or arterial embolism; (3) Lymphatic embolism or permeation; (4) Implantation by direct contact.

Cellular Metaplasia.—The original cœlomic endothelium has very varied potentialities. From it are derived the alimentary canal, the peritoneum, and the urinary and the generative organs. It is tempting to suppose that on occasions this variability persists and that peritoneum turns into endometrium. If, however, this were so one might expect to find ectopic bowel or bladder mucosa with equal frequency, but this is not the case. On the other hand, it is difficult to explain the presence of endometriomata at the umbilicus or in certain laparotomy scars on any hypothesis other than cellular metaplasia.

Blood-vessel Spread.—An endometrioma of the upper arm has been described (Novak, 1940) and, if this report is genuine, it suggests strongly that arterial embolism can occur. *Fig.* 27 shows endometrial tissue lying in the lumen of a vein, so that

venous embolism is clearly a possibility. However, spread by the blood-stream is probably very rare, for were it not so one would expect to find endometriomata in the liver and lungs far more often than is in fact the case.

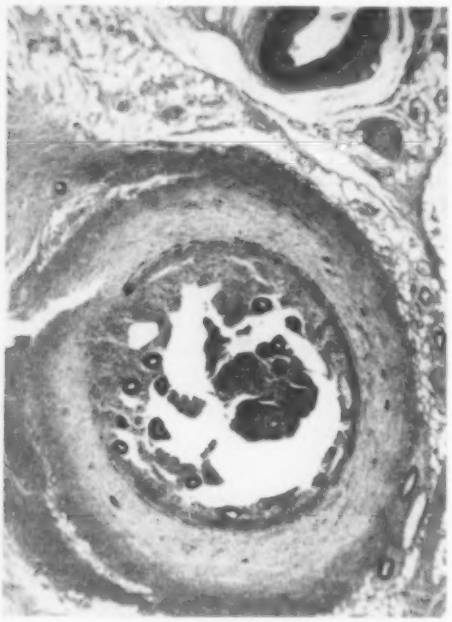


FIG. 27.—*Case* 6. Endometrial tissue lying within the lumen of a vein in the rectosigmoid mesentery. (× 30.)

Lymphatic Embolism or Permeation.—This could account for many cases of endometriosis. Endometrial tissue has been demonstrated within the lumen of lymphatic vessels but, if spread by this means is usual, it is difficult to explain the rarity with which deposits are found in lymph-nodes.

Table I.—THE DISTRIBUTION OF PELVIC ENDOMETRIOSIS (Masson, 1945)

Organ Involved	*Number*
Uterine muscle	1,852
Ovary	904
Pelvic peritoneum and cul-de-sac	511
Sigmoid, rectum, rectosigmoid	360
Fallopian tube	200
Ligaments of uterus	192
Rectovaginal septum	67
Bladder or peritoneum of bladder	62
Cervix uteri	45
Vaginal wall	44
Abdominal wall	37
Small intestine	35
Cæcum	18
Appendix	15
Umbilicus	11
Ureter	8
Femoral hernia	2
Labia	1
Vesicovaginal septum	1
Total	4365

Direct Implantation.—Sampson (1921) has been the leading protagonist of this theory and there is much evidence in favour of it. It is approximately true to say that the nearer an organ is to the uterus the more frequently is it the site of endometriosis (*Table I*). 'Retrograde menstruation' from the Fallopian tubes is often observed in the course of surgical operations, and in experimental animals endometrial tissue implanted similarly within the peritoneal cavity does grow to form endometriomata. In the present series the 3 patients with perineal endometriomata had had children respectively 5, 12, and 4 years previously. All three had suffered perineal tears during delivery and the patient whose delivery had been five years previously had had a perineal repair carried out shortly after the birth. It seems likely that endometrial cells were sown in the perineal wounds and subsequently multiplied to form endometriomata.

The most likely aetiological factors, then, seem to be direct transperitoneal implantation of endometrium by retrograde tubal menstruation, and cell metaplasia in that order of importance. Lymphatic and blood-vascular spread almost certainly do occur at times, but are probably very rare.

PATHOLOGY

The distribution of the lesions in the present series is shown in *Table II*.

The size of the lesion varied from pinhead nodules or puckered scars to masses 5 cm. in diameter.

The tumour itself consists largely of fibrous tissue (*Fig.* 28) formed as a result of repeated, cyclical hæmorrhages into its substance. Embedded in this fibrous tissue are gland tubules lined with columnar epithelium and surrounded by a characteristic stroma of loose, cellular connective tissue (*Fig.* 29). The tumour is generally in the subserous or muscle coat of the bowel, but may penetrate to the submucous layer. It is rare for the mucous membrane to be ulcerated (2 out of 17 cases) (*Fig.* 30). If the mucous membrane is involved, dark polypoid projections appear on the surface and a change in the histology occurs. Dr. Cuthbert Dukes, who was responsible for the pathological report in *Case* 20, pointed out that, although the two constituents of

FIG. 28.—*Case* 8. A mass of fibrous tissue invades the muscle coat of the bowel from the serous side. Embedded in the fibrous tissue are a few endometrial glands. Fibrous contraction has caused sharp infolding of the bowel wall on its serous aspect. (× 3.)

endometrium, the glandular tissue and loose stroma, are both easily recognizable in the muscle coat, when the growth reaches the surface only the glandular tissue may be found. This may make the interpretation of biopsies particularly difficult.

Obstruction of the bowel was demonstrable in one-third of the cases (7 out of 21). In 2 of these the size of the tumour projecting into the lumen was the obstruction can occur at a relatively early stage owing to kinking of the gut by fibrosis limited to one segment of its wall. *Fig.* 31 shows clearly this tendency to angulation.

Metastasis by the blood- or lymph-stream is so rare that its possibility can safely be ignored, but local recurrence following inadequate removal of the tumour may occur. In the present series 3 out of 3 perineal endometriomata recurred after what was thought at the time to be an adequate excision.

These tumours, then, are invasive, they tend to cause fibrosis, and are liable to local recurrence, but here the similarity to a true neoplasm ends, for an endometrioma is completely hormone dependent.

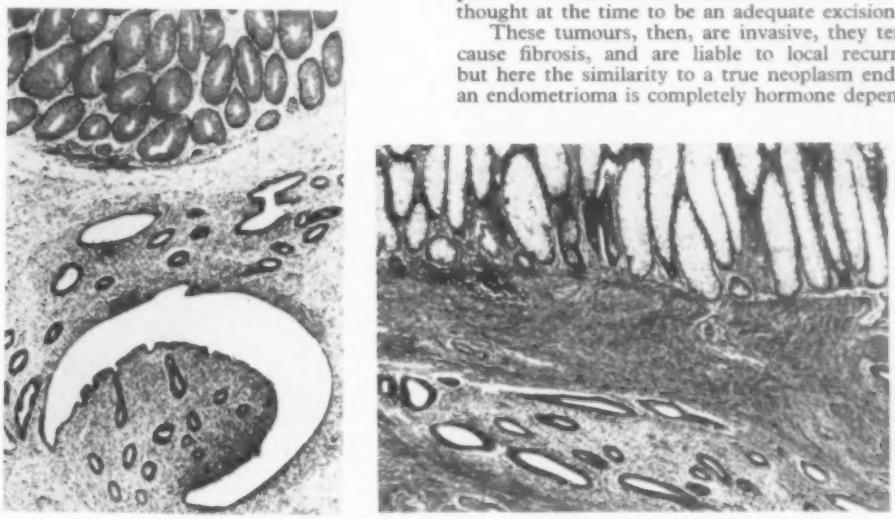
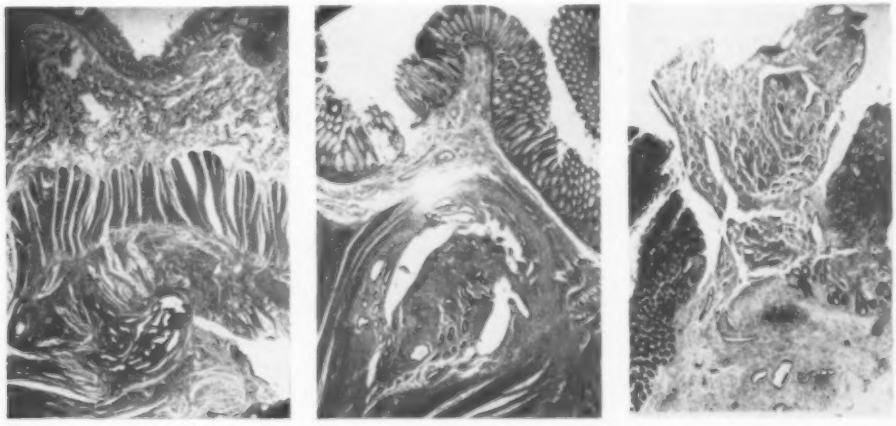


FIG. 29.—*Case* 6. Two views showing the endometrial gland tubules embedded in the characteristic stroma. Above is normal colonic mucosa. (× 37·5.)



A B C

FIG. 30.—*Case* 19. A, Endometrial tissue in the subserous coat of the bowel wall. (× 10.) B, Penetration of the endometrioma to the submucous layer. (× 10.) C, Endometrial tissue has broken through the mucous layer into the lumen of the bowel. This is rare. (× 14.)

main factor (*Fig.* 32), while in the remaining 5 a fibrous stricture in the muscle coat or kinking and fixation of the bowel by external adhesions was responsible. An endometrioma grows slowly so that the bowel is seldom completely encircled, but Cessation of ovarian function at the menopause or by castration results in regression of the tumour and disappearance of the symptoms which it has been causing, while it has also been noted that the administration of stilbœstrol to an oöphorectomized woman

has caused reactivation of a quiescent endometrioma (Faulkner and Riemenschneider, 1945). A very few cases have been reported in which the contraction of fibrous tissue has caused obstructive symptoms years after the menopause (Cattell, 1937; Colcock and Lamphier, 1950; McKittrick, 1937), and a fibrous

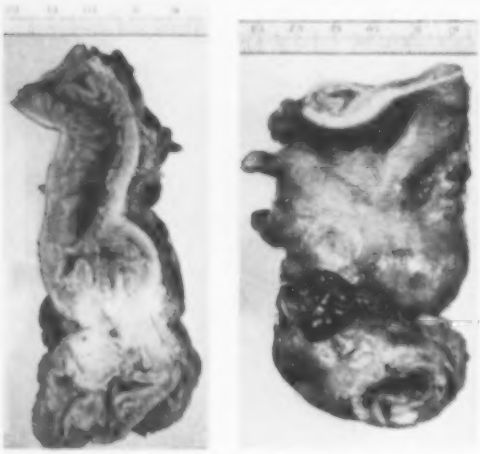


FIG. 31.—*Case* 8. An endometrioma of the sigmoid colon causing obstruction of the bowel by narrowing and angulation. (Operation specimen, Author.)

stricture already established will not of course be affected by removal of the ovaries (*Case* 13).

CLINICAL FEATURES

Patients with this disorder are most frequently in the latter half of their reproductive life, between

Table II.—THE DISTRIBUTION OF THE LESIONS IN 21 CASES OF LARGE-BOWEL ENDOMETRIOSIS

SITE	NUMBER	CASE NUMBERS
Perineum	3	1, 2, 3
Rectum	7	6, 9, 10, 12, 18, 20, 21
Sigmoid colon	9	4, 5, 8, 11, 14, 15, 16, 17, 19
Cæcum	1	7
Generalized involvement of the pelvis with fibrosis around rectum and sigmoid colon	1	13

35 and 50 years of age. The age distribution of 18 cases in this series is shown in *Table III*.

History of Gynæcological Disorder.—Of 13 patients, 1 menstruated every fourteen days, 4 had menorrhagia, and 5 dysmenorrhœa—common and unspecific complaints which were of no help in diagnosis.

Although sterility is said to be associated with pelvic endometriosis in general, the members of this series were no more than subfertile. Six, 5 of whom were married, had had no children but 8 had had 16 children between them.

Length of History.—Previous writers (Mayo and Miller, 1940, McGuff, Dockerty, Waugh, and

FIG. 32.—*Case* 6. A very large endometrioma which has spread from the uterus to penetrate to the rectal lumen. The mucosa is ulcerated and the lumen partly obstructed. (Operation specimen, Mr. W. B. Gabriel.)

Randall, 1948) have found that the history of alimentary symptoms in this disorder is usually long, the average in McGuff's series being 6½ years. The

Table III.—AGE DISTRIBUTION OF 18 CASES OF LARGE-BOWEL ENDOMETRIOMA. CASE NUMBERS

Age	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59
		1	20	3	2	13		16
		5		8	6	14		
				19	7	18		
					9			
					10			
					11			
					15			
					17			

Table IV.—THE LENGTH OF HISTORY IN 14 CASES OF LARGE-BOWEL ENDOMETRIOMA

	Case No.	*Duration of Symptoms in Months*
One year or less	1	12
	3	6
	4	8
	5	2
	6	12
	9	12
	10	8
	11	1
	12	6
	19	1
Over one year	2	24
	7	60
	8	48
	13	24

findings in the present series were directly contrary, the average length of history where the endometrioma was the sole lesion being seventeen months (*Table IV*).

Addition of the five cases in which the predominant symptom-causing lesion was a carcinoma increases the average to 18½ months.

SYMPTOMS AND SIGNS

The Perineal Lesions.—In these 3 cases the complaint was of a tender lump near the anus. In two the lesion was more troublesome at the time of menstruation. In the other the endometrioma had become infected and discharged pus from time to time. Examination revealed a tender lump—in one case two lumps—under the perineal skin. The largest was 2·0 cm. in diameter. One had a faintly blue tinge. The histological picture was in accordance with these findings—namely, blood-filled cysts, lined with columnar epithelium, embedded in a loose connective-tissue stroma. The diameter of the cysts varied from a fraction of a millimetre to 2 cm.

In none of these cases was a correct pre-operative diagnosis made.

The Abdominal Lesion.—*Table V* shows the main symptoms and the cases in which they occurred.

It is clear that, in spite of a small overlap, the cases fall into two groups: those with obstructive and those with irritative features—colic and diarrhœa.

Table V.—The Distribution of Symptoms among 10 Cases of Large-bowel Endometrioma

Symptom	*Case Numbers*
Colicky abdominal pain	4, 5, 7, 8, 11
Constipation	4, 5, 6
Vomiting or nausea	7, 8
Heavy feeling in the pelvis	5, 6
Rectal bleeding	4, 19
Diarrhœa	9, 10, 11, 13
Loss of weight	6
Gain in weight	11

The rarity with which rectal bleeding was encountered is in accordance with the findings of other workers (Cattell, 1937; Mayo and Miller, 1940). This feature is of importance in the differential diagnosis from carcinoma.

Seven out of 11 patients said that their symptoms were more severe just before or during a menstrual period, but this association, when it was present, was by no means invariable and is not as reliable a diagnostic feature as might be supposed. Its presence is important, but its absence by no means excludes the diagnosis.

Physical examination revealed no abnormality in 5 out of 11 cases; in 3 there was evidence of subacute intestinal obstruction; in 3 a hard mass presumed to be a carcinoma was felt in the rectum, while in 1 a tender swelling, lying behind the uterus, was felt through the rectal wall.

Four representative case histories are as follows :—

Case 8.—An obstructive lesion.

Mrs. H. C., aged 39 years, was admitted to hospital as an emergency under the care of Mr. V. C. Pennell.

History.—In the three years before being seen she had had four attacks of lower abdominal colic separated by intervals of about eleven months. The pain lasted for from two hours up to three days, which was the duration of the present attack. She had never vomited with the attacks, but was sometimes nauseated. The pain was associated with constipation and was relieved by the passage of fæces and flatus. Between attacks she was well, but had noticed increasing constipation over the past two years. Only one attack was at the time of a menstrual period.

She had been admitted to the same hospital six months previously for investigation of abdominal pain. She was observed for ten days on that occasion and then discharged with no definite diagnosis.

On Examination.—Visible intestinal peristalsis in abdomen. Bowel-sounds obstructive in nature. Rectum empty.

A radiograph of the abdomen showed distension of the colon, with fluid levels in the sigmoid region.

At Operation.—A tumour 4·0 cm. in diameter was found in the sigmoid colon, causing obstruction of the bowel above it. At the centre of the tumour was a tight stricture. There were three or four dark red, raised endometrial plaques under the serous coat adjacent to the tumour. The left ovary contained an endometrial cyst and there was fibrosis in the region of the left Fallopian tube and ovary. The left ovary and tube were removed. The tumour of the sigmoid colon was resected and the cut ends joined.

Recovery was uneventful.

Case 10.—*An Irritative Lesion.*

Mrs. M. U., aged 44 years (under the care of Mr. D. W. Bracey).

History.—Recurring diarrhœa for eight months. The diarrhœa occurred two days before each period. There was never any blood in the stools.

On Examination.—A hard mass was felt per rectum involving the anterior part of the bowel wall.

At Operation.—A hard tumour was found in the rectal wall apparently invading the surrounding tissues. The remaining pelvic organs were normal.

An abdominoperineal excision of the rectum was carried out.

Case 13. - *Obstructive and Irritative Symptoms.*

Mrs. D. G., aged 47 years. Admitted to hospital as an emergency under the care of Mr. P. H. R. Grey.

History.—Twenty-three-months' history of intermittent attacks of diarrhœa. Towards the end of this period the bowels were open fourteen times per day. There was mucus but no blood in the stools. During the last few months before admission the patient had noticed increasing abdominal distension.

On Examination.—The abdomen was distended. Hard fæcal masses were palpable in the left iliac fossa.

Per rectum a 'large hard neoplasm' was felt in the pouch of Douglas invading the anterior rectal wall.

At Operation.—"Endometriosis of the pouch of Douglas involving the right ovary and tube and the right side of the pelvis, completely encircling the lumen of the rectum without producing ulceration. It was not technically possible to remove the ovaries." The lumen of the rectum would only admit the tip of the little finger. A left iliac loop colostomy was made.

Case 7.—Mrs. L. H., aged 44 years (under the care of Mr. C. J. Gordon).

History.—For some months she had suffered recurring attacks of abdominal pain and distension. At first there had been a definite association between these attacks and her menstrual periods, but for two months before admission to hospital the pain and distension had remained constant.

On Examination.—She was suffering from subacute intestinal obstruction.

At Operation.—The obstruction was caused by a mass in the cæcum. This was removed by means of a right hemicolectomy.

Comment.—*Case* 8 demonstrated purely obstructive symptoms and signs. In *Case* 10 by contrast the manifestations were those of irritation of the

bowel. In *Case* 13 there was a combination of irritative and obstructive features, the latter replacing the former as fibrosis in the endometrioma progressed.

SPECIAL INVESTIGATIONS

Radiography.—Five barium enema X-ray investigations were made. In only one was the true diagnosis suspected. Relevant extracts from the reports were:—

Case 4.—"Carcinoma at iliopelvic junction."

Case 8.—"Filling defect at the lower part of the sigmoid was seen in all the films but was not visible after evacuation of the barium. The mucosa at this site was not ulcerated." No diagnosis was suggested and the very great importance of absence of mucosal ulceration was not appreciated.

Case 13.—". . . stricture at the rectosigmoid junction. The mucosa in the strictured area does not appear quite normal at the proximal end. This could be an endometrioma but carcinoma is more likely."

Case 15.—"Narrowed area in the pelvic colon. Consistent with a neoplasm, presumably a carcinoma."

Case 19.—"Obstruction in the lower pelvic colon."

Radiological investigation can be helpful in the diagnosis in obstructive cases provided that the mucosal pattern is examined and the significance of absence of ulceration at the site of obstruction appreciated.

Sigmoidoscopy.—The usual situation of an endometrioma either in the subserous or the muscular coat detracts considerably from the value of sigmoidoscopy as a diagnostic aid. Rigidity and hyperæmia of the bowel wall may be noted. On the rare occasions when the mucosa is ulcerated the appearance is very difficult to distinguish from that of a carcinoma.

Typical reports were:—

Case 4.—"Bowel appeared rigid at 13 cm."

Case 6.—"Typical carcinoma of the rectum."

Case 19.—"Considerable engorgement of the mucosa. Blood and mucus + + in lower pelvic colon."

Sigmoidoscopic Biopsy.—Unfortunately the results of this are often quite as deceptive as those of naked-eye inspection. The depth of the lesion beneath the mucosa makes it difficult to obtain a representative piece of tissue, and with superficial lesions the histological appearances can be most misleading.

Case 9.—A biopsy report read ". . . fragment of an adenocarcinoma of the rectum. Grade 2 malignancy." The report by Dr. Cuthbert Dukes on the excised specimen from the same patient read: "The part of the growth embedded in the rectal muscle and perirectal fat has the typical histology of an endometrioma, but, where the tumour has spread into the rectal mucous membrane and submucosa, the histological appearances are different. Here, on the surface, the tumour consists of proliferating columnar epithelium showing an irregular glandular arrangement and exactly resembling rectal cancer of an average grade of malignancy."

Case 6.—The report was "low-grade adenocarcinoma".

Case 21.—This was the only successful diagnosis.

First biopsy: ". . . areas which are suspicious but not absolutely characteristic of malignancy".

Second biopsy: "The sections show a small nodule of endometrioma lying in the rectal muscle."

Where the pathologist is in doubt, and, if it is possible that the lesion may be an endometrioma, a further deep biopsy should be taken in order to obtain a piece of the muscle wall of the bowel.

The disease being so rare and the diagnostic pitfalls so numerous, it is perhaps not surprising that in this series the pre-operative diagnosis in 15 out of 16 cases was carcinoma.

TREATMENT

The Perineal Lesions.—Two of these were excised and the skin sutured. There was almost immediate local recurrence in both cases. In one this was not troublesome enough to warrant further treatment, but in the other a second operation was needed. The lump, with its overlying skin, was excised and the wound left open. Fifteen days later the granulating surface was cauterized with diathermy. This was followed by complete healing without recurrence.

In the third case the lesion was excised and the wound left open. The skin healed, but there was recurrence of slight pain and swelling at intervals until the menopause, after which the symptoms disappeared.

Wide local excision combined with the application of thermal cautery, and without suturing of the skin, appears to be the most certain way to ensure complete destruction of the aberrant endometrial tissue in this type of case.

The Abdominal Lesions.—Accurate pre-operative diagnosis being such a rarity, it becomes important to interpret correctly the findings at operation. This was done in 5 out of 11 cases. In 5 others the lesion was thought to be a carcinoma and in 1 case no diagnosis was made.

The growth itself appears either as a hard tumour in the wall of the bowel or as a string-like stricture. It is in both cases almost indistinguishable from a carcinoma. A correct diagnosis depends on the recognition of associated pelvic endometriosis. Where the characteristic small red plaques of endometriosis are visible in the subserous coat of the bowel adjacent to the tumour there is little difficulty. The lesions may, however, be more distant, either 'chocolate cysts' in the ovaries or scattered peritoneal endometriomata. It is then less obvious that these lesions are related to that in the bowel wall and, moreover, the experience from this series shows that a bowel carcinoma may well be associated with pelvic endometriosis.

Peritoneal endometriosis itself may be difficult to identify when in a quiescent phase. It appears then as a patch of subserous fibrosis with puckering of the peritoneum over it or simply as a fibrous adhesion. The latter can very easily be mistaken for malignant infiltration (*Case* 6).

As at sigmoidoscopy, biopsy may be misleading. An operation biopsy taken from one endometrioma in the present series was thought to be a fully representative specimen, but was reported: "A piece of muscle, no evidence of carcinoma or endometriosis." In cases of doubt, where microscopy is required, the fragment removed must include the full thickness of the bowel wall.

Table VI shows the operative findings and the operative diagnoses of the cases in this series.

There appears to be a reasonable chance of recognizing the lesion at operation if its possibility

is kept in mind and if a careful search is made for associated pelvic endometriosis. Nevertheless in 6 out of 10 with abdominal lesions in this series the tumour in the bowel was solitary and very difficult to distinguish from a carcinoma.

The aim of treatment should be to remove the bowel lesion and at the same time preserve the ovaries. In some cases the ovaries themselves are affected and require removal for that reason, but if one or both are normal they or it should be spared. An exception to this generalization is a case where removal of the bowel lesion can only be achieved at the expense of a permanent colostomy. Most patients would prefer an early menopause to this.

Fig. 32 (*Case* 6) shows such a case which, had it been diagnosed either at or before operation, could probably have been adequately treated by ovariectomy. Where obstruction is present a temporary proximal colostomy may be necessary whatever final treatment is decided upon.

Table VII summarizes the treatment and results in the present series. In 4 out of 11 cases (*Cases* 5, 7, 8, 19) the endometrioma was resected, bowel continuity restored, and ovarian function left undisturbed. In none of these has there been any recurrence. In 3 cases (*Cases* 6, 9, 10) the rectum was excised completely and one or both ovaries spared. Assuming the technical impossibility of restoring continuity of the bowel, a preferable treatment in these cases would have been oöphorectomy, with or without a temporary colostomy.

Table VI.—The Operative Findings and Diagnoses in 13 Cases of Large-bowel Endometriosis

Case	Findings	Operation Diagnosis
1	Cystic swelling in perineum containing altered blood	?
2	Subcutaneous fistula between vulva and perineal skin	?
3	Hard nodule in perineum	Endometrioma
4	Indurated stricture of pelvic colon with two small deposits of peritoneal endometriosis in relation to it	Endometrioma
5	Nodular growth of pelvic colon adherent to left ovary	Carcinoma
6	Mass in rectosigmoid adherent to uterus	Carcinoma
7	Mass in right iliac fossa involving terminal ileum and cæcum	?
8	Firm tumour of sigmoid colon with tight stricture at its centre. Subserous endometrial plaques on bowel adjacent to the tumour	Endometrioma
10	Hard tumour in wall of rectum apparently invading surrounding tissues. No other abnormality in pelvis	Carcinoma
11	Large hard mass at pelvirectal junction. Endometriosis of both ovaries	Carcinoma
13	Generalized fibrosis in pelvis involving rectum and ovaries	Endometriosis
15	Mass 1 in. in diameter in the wall of the sigmoid colon occupying two-thirds of its circumference	? Endometrioma ? Carcinoma Biopsy
19	Growth just above pelvirectal junction. Uterus and ovaries normal	Carcinoma

Three cases (*Cases* 4, 11, 15) had a resection and anastomosis with oöphorectomy. In one of these both ovaries contained endometriomata, but in the other two their removal was unnecessary.

In 1 case (*Case* 13) general pelvic endometriosis caused a stricture of the rectum by fibrosis. Owing to dense pelvic adhesions it was not possible to remove the ovaries. A proximal colostomy was made and a castration dose of X rays subsequently delivered to the pelvis. In spite of this the fibrous stricture remained and this colostomy is regarded as permanent.

Table VII.—Treatment and Results in 11 Cases of Endometriosis of the Colon and Rectum

Case	Operative Treatment	Oöphorectomy	Follow-up
4	Resection of tumour at iliopelvic junction and anastomosis of cut ends of bowel	Yes	No further trouble
5	Paul-Mikulicz resection of part of pelvic colon. Continuity of bowel restored	No	Daughter born 2 years later. Hysterectomy 10 years later for metropathia hæmorrhagica. No sign of recurrence of endometriosis
6	Perineo-abdominal excision of rectum	No	One year later 'moderate' course of X-ray therapy to pelvis. Health very good 7 years later at age 56
7	Right hemicolectomy with iliotransverse anastomosis	No	Four years later still menstruating. Very well. No symptoms
8	Resection of tumour in pelvic colon with anastomosis	No	One year later, very well and symptom-free
9	Synchronous combined excision of rectum	No	Eight years later, perfectly well
10	Abdominoperineal excision of rectum	No	Perfectly well 1 year later. Referred for radium menopause, but this was thought to be unnecessary and was not carried out
11	Resection of tumour in rectosigmoid with anastomosis	Yes	One year later, very well but complained of loss of sexual desire
13	Sigmoid colostomy for generalized pelvic fibrosis	No	One month later a radiation menopause was induced. Four years later, general health good, periods ceased, colostomy satisfactory
15	Resection of tumour of pelvic colon with anastomosis	Yes	No further symptoms
19	Resection of tumour of pelvic colon with anastomosis	No	Ten years later, perfectly well

There were no operative deaths among the patients with endometriosis alone. One patient (*Case* 12), who had a carcinoma of the rectum as well as an endometrioma higher up in the bowel, died following abdominoperineal excision of the rectum.

SUMMARY

Endometriosis of the large bowel is rare, very much rarer than carcinoma.

Twenty-one cases are reported. Of these lesions 3 were in the perineum.

It is possible that 1–2 per cent of all women develop endometriosis of the large bowel during their menstrual life, but the great majority of these lesions must be symptomless.

Direct transperitoneal implantation and cellular metaplasia are probably the most important aetiological factors. The size of the lesions varies from pinhead nodules to tumours 5 cm. in diameter. The lesion is usually situated in the subserous or muscular coat of the bowel. The main bulk of the tumour is made up of fibrous tissue, contraction of which may cause obstruction of the bowel.

When the lesion penetrates to the mucous or submucous layer of the bowel its histological appearance comes to resemble that of carcinoma very closely. The tumour is completely hormone dependent.

Patients are usually between 35 and 50 years of age. The average length of history was seventeen months. The perineal lesions presented as tender nodules under the skin. Two out of three were more troublesome at the time of menstruation.

The symptoms from the abdominal lesions are either irritative or obstructive. Rectal bleeding is rare. Worsening of symptoms at menstruation may occur, but is not invariable. Physical examination may reveal no abnormality at all; a hard tumour may be felt in the rectum or there may be signs of subacute intestinal obstruction.

Diagnosis by barium enema depends on recognition of the absence of mucosal ulceration at the site of the lesion.

The appearances at sigmoidoscopy are difficult to distinguish from carcinoma, while biopsy specimens may also resemble carcinoma very closely. The taking of a deep bite of tissue, to include the muscle coat, may be necessary if a correct diagnosis is to be made. A correct pre-operative diagnosis was made in 1 out of 16 cases.

Destruction of the perineal lesions is most certainly achieved by excision, cautery to the wound, and allowing the wound to heal by granulation.

Five out of 11 abdominal cases were diagnosed at operation. Diagnosis depends on the recognition of associated lesions of endometriosis, particularly the identification of raised red subserous deposits adjacent to the tumour.

If possible the lesion should be excised, bowel continuity restored, and one or both ovaries preserved.

If excision of the endometrioma can only be achieved at the price of a permanent colostomy, bilateral oöphorectomy is likely to cure the disease and is a preferable treatment.

In cases where generalized pelvic fibrosis is very extensive a permanent colostomy may be necessary.

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AORTO-POPLITEAL GRAFTS FOR ATHEROSCLEROTIC OCCLUSION

BY T. S. MATHESON AND H. S. SHUCKSMITH

THE GENERAL INFIRMARY AT LEEDS

REPORT OF TWO PATIENTS

WHEN the distal aorta, the iliac arteries, and the femoral arteries are blocked by atherosclerosis, a by-pass graft from the aorta, to improve the blood-flow to a lower limb, can be carried out with success if the popliteal artery be patent. Linton (1954), Linton and Menendez (1955), and Crawford, Creech, Cooley, and De Bakey (1955) have reported examples of the use of long homografts and we present 2 patients in whom aorto-popliteal grafts have been used.

CASE REPORTS

Case 1.—J. C., male, aged 61 years. Blood-pressure 200/100 mm. Hg. There was a seven-year history of intermittent claudication which was relieved by bilateral lumbar sympathectomy for two years, but was slowly progressing over the last year. Finally the claudication became so severe that pain in the right calf and thigh stopped him from walking after 50 yd. and pain in the right foot disturbed his sleep. At this time, three years after sympathectomy, the left common femoral pulse was present but the right common femoral pulse was absent, and there were nutritional changes in the toes of the right foot.

Translumbar aortography showed complete obstruction of the right common and external iliac arteries, patency of the left iliac arteries, and poor filling of both superficial femoral arteries (*Fig.* 33). The aortogram and arteriography by open dissection of the right femoral artery showed good filling of the right popliteal artery by collateral vessels (*Figs.* 34, 35).

A freeze-dried arterial homograft was inserted from the lower part of the right popliteal artery to the aorta, which had been divided at its termination at the right

common iliac artery. The anastomosis to the popliteal artery was carried out with the patient in the lateral position, and this graft was tunnelled up to the femoral triangle before the patient was turned on to his back for the abdominal part of the operation. An aortic bifurcation homograft was used and the long limb divided. The distal portion was passed up from the popliteal fossa to the femoral triangle, and the proximal portion was passed downwards under the inguinal ligament to the femoral triangle, and at this level the two portions were anastomosed together. The bulk of the undivided graft made upward tunnelling through an œsophagoscope and passage under the inguinal ligament impossible. The operation restored the right dorsalis pedis pulse and it has remained easily palpable for one year.

FIG. 33.—*Case* 1. Aortogram showing blockage of the right iliac arteries.

Case 2.—T. C., male, aged 52 years. Blood-pressure 220/120 mm. Hg. This patient had suffered from intermittent claudication for three years and pain in the left calf stopped him walking after 30 yd. on flat ground. The right common femoral pulse was present, but the left common femoral pulse was absent and early ischæmic changes were seen in the toes of the left foot.

Translumbar aortography showed a partly blocked bifurcation of the aorta, complete obstruction of the left external iliac artery, and irregularity of the right iliac arteries (*Fig.* 36). Both superficial femoral arteries were blocked, but good filling of the popliteal arteries had been re-established by collateral vessels (*Fig.* 37).

An aortic bifurcation homograft was inserted from the divided lower aorta to the right common femoral artery and, on the left side, it was anastomosed in the femoral triangle to a second homograft which had already been joined to the popliteal artery. The distal pulses in the lower limbs were restored, but seventeen days after operation thrombosis occurred and blocked the left limb of the graft. The lower limb survived this misfortune.

Nine months after the original operation a Teflon graft was inserted from the aortic portion of the homograft to the left popliteal artery, distal to the original anastomosis. Ankle pulses were restored and have remained palpable for the subsequent nine months' follow-up period.

FIG. 34.—*Case* 1. Aortogram with tourniquet on left thigh. There is good filling of the right popliteal artery by collateral circulation.

FIG. 35.—*Case* 1. A well-filled but narrow right popliteal artery on right femoral arteriography.

Pertinent Operative Details.—The popliteal arteries were exposed with the patients on their sides; the upper leg rested on a stool with the knee extended. If the popliteal artery was felt to be pliable a 1–1·5 cm.

longitudinal incision was made in the vessel to establish the presence of a good backflow of blood. A polythene catheter was introduced distally into the popliteal artery and the limb infused with 20 ml. of heparin in normal saline (100 mg. per litre). A femoropopliteal homograft was tunnelled from an incision in the femoral triangle over the femoral artery through the adductor canal to the popliteal fossa and the lower anastomosis carried out. In the first patient Mavor's (1956) technique of anastomosis was used; the end of the graft was divided obliquely and a continuous everting stitch applied. Cockett's method of anastomosis, described by Linton and Menendez (1955), was employed in the popliteal fossa for the second patient. Before the abdominal part of the operation the graft was distended with heparin in saline solution to show any gross leakage of this anastomosis.

In the first patient the right common iliac artery was divided flush with the aorta and 1 in. of common iliac artery excised and a ligature placed on the distal end of this artery because of bleeding. The iliac portion of the graft was passed retroperitoneally and under the inguinal ligament to the incision in the femoral triangle and was then anastomosed to the aorta and to the distal graft. The proximal graft was washed out with blood and the distal graft was filled with heparin in saline solution before the ends of the two grafts were joined by end-to-end anastomosis. In the second patient the aorta was divided 1½ in. above its bifurcation and an aortic bifurcation homograft sutured to the aorta. The end of the right limb of the graft was sutured to the side of the right common femoral artery and, on the left, it was joined end-to-end in the femoral triangle to the graft which had been anastomosed to the left popliteal artery already. A tear in the middle of the femoral portion of this graft necessitated its exposure and a further end-to-end anastomosis of this part of the graft.

DISCUSSION

By-pass grafts in general give favourable results in the larger arteries, because the lumen of an artery distal to a block caused by atherosclerosis is commonly patent, particularly when the block is complete, e.g., the common femoral artery in aorto-iliac obstruction and the distal portion of the popliteal artery in blockage of the superficial femoral and upper part of the popliteal arteries. Arteriography is most useful in showing patency, but when the obstruction is extensive, as in aorto-iliofemoral occlusion, the collateral vessels may not carry enough contrast medium from the translumbar aortic injection to fill the lumen of the popliteal artery to its limit. Conversely, because arteriograms only show the anteroposterior view, the popliteal artery in particular may appear to have a much larger lumen than in fact exists. Atherosclerosis nearly always involves the anterior wall of the popliteal artery more than the other walls and the atheroma may project into the lumen in a mushroom fashion so that the lumen, while appearing wide on the arteriogram, is only a crescentic slit (*Fig.* 38). Whatever the arteriogram may show, whether the medium has been introduced by the translumbar aortic route or by Seldinger's (1953) percutaneous technique of femoral arteriography, the final opinion on the state of the lumen of the popliteal artery depends on inspection of the artery after incision of its wall. The first part of the operation for aorto-popliteal by-pass grafting is, therefore, the exposure of the popliteal artery.

In both patients the approach to the popliteal artery was according to the description of Owen and Rob (1956). The operation was performed with the patient in the lateral position and the graft was

FIG. 36.—*Case* 2. Aortogram showing a narrowed aortic bifurcation, a blocked left external iliac artery, and apparently well-filled right common and external iliac arteries.

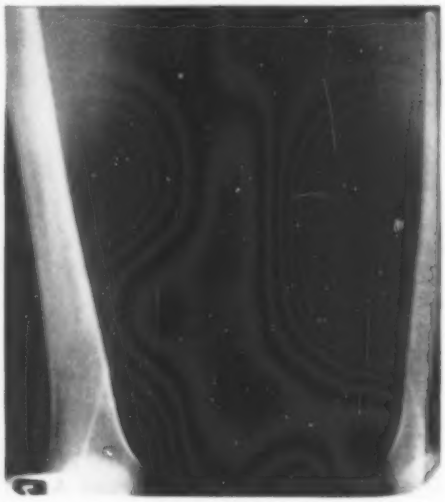

FIG. 37.—*Case* 2. Good filling of the popliteal arteries on aortography is seen above each knee.

anastomosed to the popliteal artery before turning the patient on to his back for exposure of the abdominal aorta. It was necessary to divide the long homografts and to join them again by end-to-end anastomosis in femoral triangles. Exposure of the popliteal artery by Henry's (1945) approach enables the whole operation to be carried out with the patient supine.

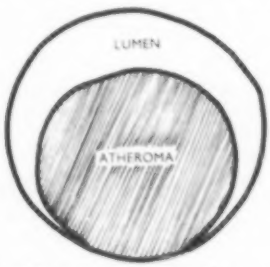

FIG. 38.—Schematic cross-section of an artery mostly occupied by an atheromatous plaque, but which may show good filling on arteriography.

The aortic bifurcation and the popliteal artery are exposed and the condition of these vessels is assessed. The long limb of an aortic bifurcation graft is passed retroperitoneally under the inguinal ligament and then to the popliteal fossa before any anastomosis is made. The supine position allowing the passage of the long limb of the graft the whole way downwards makes one less anastomosis necessary.

The second patient was unfortunate to develop a venous thrombosis in his left leg on the fourteenth post-operative day. Dindevan was given in sufficient dosage to maintain a prothrombin time of 40 sec., yet on the seventeenth day after operation thrombosis developed in the long limb of his graft in the same leg. The limb survived this hazard, which may be attributable to the presence of two end-to-end anastomoses in the course of the graft in the thigh.

The progress of atherosclerosis cannot be halted and the use of long arterial grafts is desirable in a selected number of patients. Even with a significant mortality from cardiac, cerebral, and renal causes in these individuals and a failure of a certain number of grafts which thrombose immediately or after an interval, the insertion of such grafts is worthwhile to alleviate the claudication, rest pain, and ischæmic skin lesions from which they suffer.

SUMMARY

1. A small number of aortopopliteal arterial homografts have been reported in cases of extensive atherosclerotic occlusion.

2. Two patients with homografts from the aorta to the popliteal artery are reported and some of the operative details are briefly described.

3. The difficulties of assessing the state of the lumen of the popliteal artery by arteriography are discussed and its condition can only be decided definitely at operation.

4. Divided homografts were used because the popliteal artery was exposed with the patient on one side; the supine position is now advocated.

5. Adequate dosage of Dindevan did not prevent thrombosis developing in one graft seventeen days after operation.

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RECURRENT INTUSSUSCEPTION IN AN ADULT, DUE TO INTESTINAL METASTASES OF A BREAST SARCOMA

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INTUSSUSCEPTION in an adult is but rarely seen in a surgical lifetime. Brown and Michels (1952) found that during a period in which 430,000 new patients were registered at the Cleveland Clinic only 15 cases of adult intussusception were recorded. At the Albany (N.Y.) Hospital, in the period 1921–47, Donhauser and Kelly (1950) found that of 242,829 patients, only 12 cases of intussusception in adults were recorded.

When intussusception in an adult does occur it is axiomatic that in the overwhelming majority of instances the presence of a tumour or, more rarely, some other localized lesion of the intestinal wall must be assumed to be the underlying cause.

Recurrent intussusception in an adult is rarer still, and is often due to the causative tumour not being recognized when the intussusception is first reduced. Oughterson and Cheever (1929) quoted 12 such cases collected from the literature, and added a case of their own, where the causative tumour

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passed unrecognized at the first laparotomy, and recurrent intussusception occurred. In all the 13 cases quoted, the recurrent intussusceptions were due to primary tumours of the bowel.

FIG. 39.—Radiograph of jejunum following a barium meal, showing the transversely striated shadow formed by the intussuscipiens.

FIG. 40.—The loop of jejunum resected at the first laparotomy showing the intussusception. (× ⅔.)

The present case is recorded for the following reasons.

Recurrent jejunojejunal intussusception occurred, not because multiple tumours in the jejunum were overlooked at the first operation, but because rapidly growing new tumours appeared in the jejunum in the three months between the first and second intussusceptions.

The tumours were secondaries from a sarcoma of the breast, which was not recognized as such when removed nearly four years previously. Willis (1952), in his survey of secondary tumours in the intestine, does not mention sarcoma of the breast among the primary sites responsible, either in his own series or among the published cases.

The authors have been unable to trace any record of a similar case.

This case draws attention to the malignant potentialities of the giant fibro-adenoma, or 'cystosarcoma phylloides', of the breast, a tumour that is sometimes considered to be in most instances benign.

CASE REPORT

Mrs. E. M., aged 66 years, had borne one child. She was admitted to hospital on Jan. 22, 1958, with a history of attacks of upper abdominal colicky pain for two months and melæna for three weeks. For some weeks prior to admission she had vomited on several occasions when the bouts of abdominal pain occurred. The vomitus contained bile but never blood. She had always been in good health apart from an operation for removal of the left breast on May 24, 1954.

ON EXAMINATION.—The patient was found to be pale and obviously anæmic. The left breast had been removed totally and the right breast was normal. No peripheral lymph-glands were palpable. The abdomen was obese and difficult to examine. Apart from slight epigastric tenderness no other abnormal signs were discovered in the abdomen. Auscultation of the chest revealed signs of consolidation at the right base. Rectal examination revealed the presence of melæna.

Laboratory Findings.—Hæmoglobin was 6·2 g. per 100 ml., white blood-count 40,000 per c.mm., 80 per cent polymorphonuclears. Non-protein nitrogen was 31 mg. per 100 ml. Serum electrolytes were within normal limits. Transfusion with 5 pints of blood raised the hæmoglobin to 9·2 g. per 100 ml.

Radiological Examination.—Plain radiographs of the abdomen on the day following admission revealed no abnormality. There were no fluid levels present. Chest radiography revealed "pneumonic consolidation in the right lower lobe". A barium meal and follow-through examination performed on Jan. 16, 1958, before admission, revealed a normal stomach and duodenum. The first loop of the jejunum was enormously distended, however, and was superimposed on a feathery pattern suggestive of an intussusception, the distended transversely striated shadow representing the intussuscipiens (*Fig.* 39).

FIG. 41.—The lower specimen shows the polypoid tumour found at the apex of the intussusception at the first laparotomy; the upper specimen shows part of the jejunum resected at the second laparotomy, with a very similar polypoid tumour, again at the apex of the intussusception and a smaller tumour just distal to it. (× ⅔.)

AT OPERATION.—The first laparotomy was performed (P. G. C.) on Feb. 6, 1958, through a right upper paramedian incision. A jejunojejunal intussusception was discovered within 6 in. (15 cm.) from the duodenojejunal

flexure. A tumour could be palpated at the apex of the intussusception. There was blood in the small bowel below the lesion. No other abnormality was discovered in the abdomen or pelvis, and careful palpation of the remainder of the intestinal tract revealed no further tumours. The liver was normal to palpation. An attempt was made to reduce the intussusception, but as the bowel wall began to split, this attempt was abandoned, and 8 in. (20 cm.) of jejunum, including the intussusception, were resected and an end-to-end anastomosis was fashioned. The resected segment of jejunum is shown in *Fig.* 40.

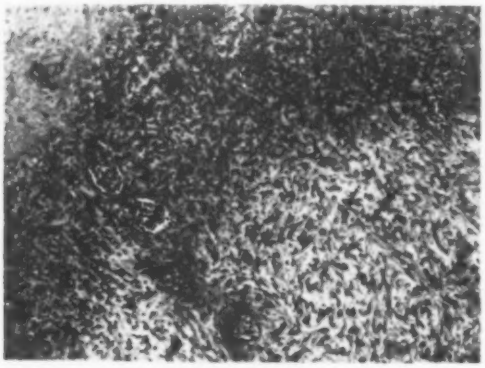

FIG. 42.—Granulation tissue covering the surface layers of the sarcomatous tumour in the jejunum. (× 80.)

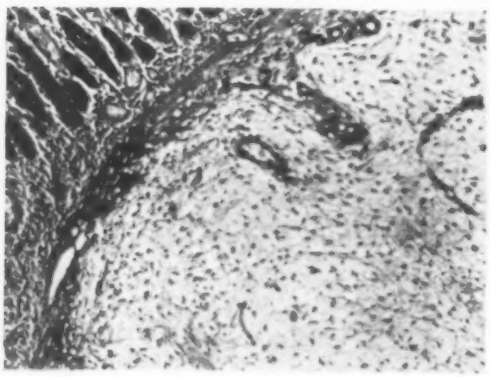

FIG. 44.—Tumour composed of small stellate cells in a mucinous stroma, situated in the submucosa of the jejunum. (× 70.)

The post-operative course was uneventful and the patient was discharged from hospital, with a hæmoglobin of 12·8 g. per 100 ml., on Feb. 24, 1958.

PATHOLOGY.—

Naked-eye.—A segment of small intestine, 13 cm. long. Near one end was a short intussusception (*Fig.* 40) and at its apex there was a pedunculated cauliflower-shaped tumour, 10 × 6 × 4 cm., which filled and distended the gut. The tumour surface was nodular, and when bisected was seen to consist of fairly firm white tissue, with a greenish tinge in places; the tumour appeared to be arising from the submucosa at the apex of the intussusceptum (*Fig.* 41). At the proximal margin of the specimen, a second polypoid tumour, 0·7 cm. in diameter, projected into the lumen.

Histology.—The large tumour is of connective-tissue origin. It shows spindle-shaped cells resembling fibroblasts in some areas, and a looser myxoma-like pattern of stellate cells in others. The surface shows granulation tissue and fibrinous exudate. In the deeper parts of the

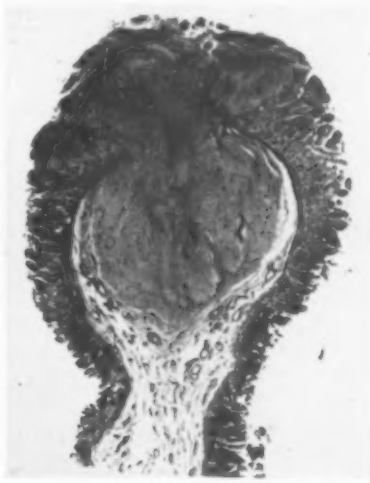

FIG. 43.—Small myxoma-like tumour, arising in the submucosa of the jejunum, and destroying the muscularis mucosæ and overlying mucosa. (× 9.)

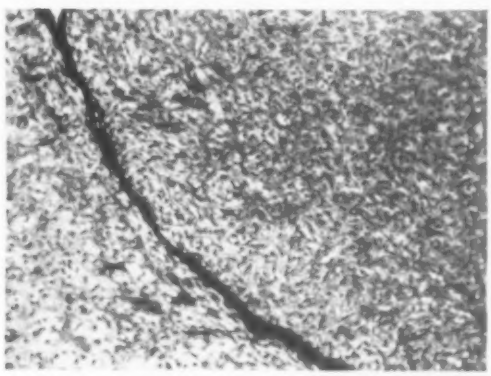

FIG. 45.—Section of breast tumour, showing a narrow epithelial cleft and the cellular stroma. (× 70.)

tumour especially, the cellularity, the variation in cell size, and the large number of mitoses indicate that this tumour is sarcomatous (*Fig.* 42). It appears to have arisen from the muscle or connective tissue of the bowel, but it shows no evidence of muscle-fibre differentiation and it was considered to be a fibrosarcoma. The small polypus has the pattern of a myxoma; it has breached the muscularis mucosæ (*Fig.* 43). It is not obviously malignant histologically, but its presence raised the possibility of further tumours elsewhere in the intestine.

READMISSION.—The patient was seen again at an out-patient clinic on April 29, 1958, complaining of recurrent upper abdominal colicky pain and vomiting of bile-coloured fluid over the previous two weeks. She had also lost weight—about 14 lb. (6·4 kg.)—since her discharge from hospital, and was again anæmic (Hb 11·3 g.

per 100 ml.). On palpating her abdomen a large sausage-shaped swelling was felt to the left of the umbilicus.

A diagnosis of recurrent small-bowel intussusception was made and the patient was admitted to hospital.

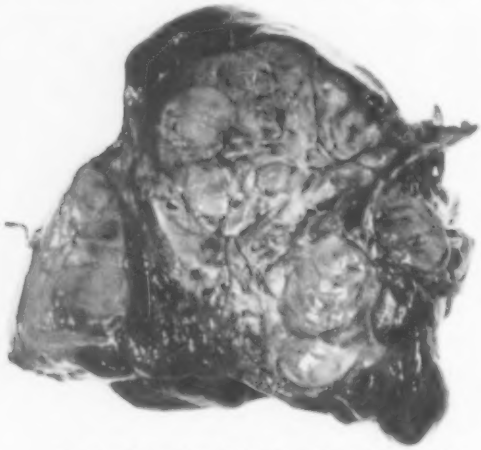

FIG. 46.—Cut surface of right lung (lower lobe) showing a large mass of soft fleshy tumour, projecting at one point into the lobar bronchus.

FIG. 47.—Medial aspect of right lung. A polypoid tumour mass is seen lying in the inferior pulmonary vein.

Clinical examination also revealed a solid tumour about the size of a golf-ball in the left temporal region of the scalp. Auscultation of the chest demonstrated signs of consolidation of the lower parts of the right lung. No further investigations were made because of the patient's poor general condition. Two pints of blood were transfused before operation.

SECOND OPERATION.—The second laparotomy (P. G. C.) was performed on May 5, 1958. The old right paramedian scar was excised and the abdomen opened. An intussusception was found in almost the same site as previously, close to the duodenojejunal flexure. Several small tumours could be felt in the jejunum on this occasion, the most proximal being only 2 in. (5 cm.) from the duodenojejunal flexure and the most distal 2 ft. (60 cm.) farther along the jejunum. About 2 ft. 6 in. (75 cm.) of jejunum were resected, including all palpable tumours, and an end-to-end anastomosis was performed. There were no metastases palpable in the liver and there was no obvious lesion in the alimentary tract distal to the involved segment of jejunum, apart from a globular distended appendix, which was removed. The tumour of the scalp was also excised. On neither of the two occasions when the bowel was resected were any mesenteric glands palpable.

PATHOLOGY.—

Naked-eye.—Examination of the jejunum revealed a 75-cm. length of small intestine, with an intussusception 15 cm. from one end, and containing four separate tumours. The largest, lying at the apex of the intussusception, was a soft fleshy polypoid mass, 5 cm. in diameter. Two smaller tumours lay within 5 cm. of the large one; one was situated in the submucosa, the other projected into the lumen as a pedunculated mass. The fourth tumour was a tiny nodule in the submucosa of the bowel some 50 cm. distal to the other tumours. *Fig.* 41 shows the very similar polypoid tumours resected on the two separate occasions from the jejunum.

FIG. 48.—A soft sausage-shaped tumour lying in the œsophagus. It is attached to the wall by a short pedicle at its upper end. (× ⅔.)

Histology.—All the *jejunal tumours* are very similar to each other, and to the tumours resected previously. They consist of pleomorphic spindle-cells with, in places, collagen bundle formation; the tiny isolated nodule has, however, more the pattern of a myxosarcoma, with stellate cells embedded in a mucin-rich matrix (*Fig.* 44). The smaller tumours have clearly developed in the submucosa of the intestine and show varying degrees of invasion and destruction of both the mucosa and the underlying muscle coats.

FIG. 49.—Secondary tumours in left parietal lobe (*top left*), right occipital lobe (*top right*), and cerebellum (*below*). (× 1.)

Appendix—a mucocele; no evidence of tumour.

The scalp tumour was a dome-shaped mass, 5 cm. in diameter, covered by intact skin; cutting revealed a soft, almost gelatinous tumour. Histologically, this growth repeats the microscopical pattern of the jejunal tumours and is clearly a metastatic sarcoma.

The jejunal tumours at the first laparotomy were thought to be either multifocal primary sarcomas of the intestine or, possibly, secondaries from an unknown primary. At this time clinical opinion had not connected the 'fibro-adenoma' removed in 1954 with the tumours of the small bowel, and the pathologists were not then aware of the history of a previous mastectomy. With the removal of the scalp tumour it became quite obvious that there was wide blood-borne tumour dissemination, and at this stage the previous history of a breast tumour came to light. The material available from the mastectomy performed in 1954 was now re-examined.

Pathology of Breast Tumour removed in 1954.—The breast was described as containing a firm, fibrous, and apparently encapsulated tumour, 2½ × 1½ in. (6 · 3·5 cm.), lying above the nipple. The histological report made in 1954 indicated that this was a large intracanalicular fibro-adenoma, with a very great amount of fibrous tissue, but showing no evidence of malignancy.

A review of the sections confirmed the presence of a large fibro-adenoma; the marked overgrowth of the fibrous component and the rather coarse club-like arrangement of the intracanalicular projections indicate that this tumour is a 'cystosarcoma phylloides' (giant fibro-adenoma, or intracanalicular myxoma). It is often extremely difficult to assess the malignancy of these tumours, but parts of the fibrous tissue component of this tumour show a degree of cellularity and pleomorphism suggestive of malignant change (*Fig.* 45). There is little doubt that the intestinal tumours are, in fact, metastases from the sarcomatous component of this mixed tumour of the breast.

Progress.—The patient's post-operative recovery was slow, and on May 17, 1958, a chest radiograph revealed a dense shadow in the region of the right lower lobe, consistent with a large secondary deposit in the lung.

The patient's condition deteriorated steadily, and she became comatose; during the terminal twelve hours her temperature reached 105° F.; she died after a period of 48-hours' coma on May 30, 1958.

POST-MORTEM EXAMINATION.—The relevant post-mortem findings can be summarized as follows.

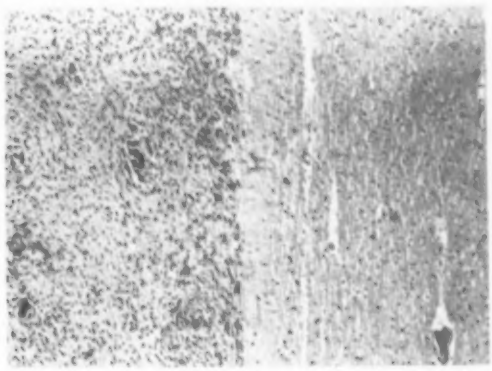

FIG. 50.—Section of secondary deposit in cerebrum, showing the sarcomatous tumour and its sharp line of demarcation from the surrounding white matter. (× 70.)

External Appearances.—An emaciated and anæmic elderly woman. Healing upper abdominal incision. No recurrence of scalp tumour and no skin secondaries seen elsewhere.

Lungs.—The right lung showed a large mass of soft fleshy white tumour occupying the greater part of the middle and lower lobes (*Fig.* 46) and forming nodular masses beneath the pleura. A small ragged piece of tumour projected into the main lower lobe bronchus. The tumour had invaded the pulmonary veins, and a large rounded tumour mass projected from the right inferior pulmonary vein (*Fig.* 47).

Alimentary Tract.—The œsophagus contained a pedunculated sausage-shaped tumour, attached by a short narrow pedicle to the mucosa of the anterior wall of the œsophagus. This tumour was very soft, and had apparently caused no obstruction (*Fig.* 48).

The *duodenum* and *jejunum* contained multiple small polypoid tumours, similar to those in the resected intestine.

Brain.—A soft, rounded tumour mass lay in the cerebellum, distending and obstructing the fourth ventricle and causing compression and distortion of the pons. The tumour was demarcated and shelled out easily from the brain. Two smaller soft grey-white translucent tumours were present in the right occipital lobe, and one in the left parietal lobe (*Fig.* 49).

Other Organs.—Small tumour deposits were found in the liver, pancreas, both kidneys, both adrenals, and the thyroid gland.

All the tumours found at necropsy were of similar appearance and consistency: soft, greyish-white, almost translucent growths, resembling myxomas, and the cut surfaces rather gelatinous and shiny. No evidence of

recurrence was seen at the mastectomy site; the right breast showed no lesion. No lymph-gland metastases were found, either in the axilla or elsewhere.

Histology.—Sections of all the tumour deposits show once more the pattern of a rather sparsely cellular spindle-cell sarcoma with areas of more stellate-cell growth in an abundant mucinous matrix (*Fig.* 50). None of the tumours shows any epithelial elements.

DISCUSSION

Secondary neoplastic disease of the intestine is not a rare finding. Willis (1952), in a series of 500 consecutive necropsies on cases of malignant disease, found blood-borne metastases in the intestine in 10 cases (2 per cent); he was also able to collect 135 published cases of metastatic growths in the intestines. He points out that the growths almost always appear first in the submucosa; later, the tumour may form a pedunculated mass projecting into the lumen, or can cause traction of the muscle coat, which may be the starting-point of an intussusception. Willis illustrates a case of his own where a pedunculated metastasis from a breast carcinoma formed the apex of the intussusception.

When metastatic tumours of the intestine are found, multiple blood-borne metastases are almost invariably also present (Willis). In the present case, signs of consolidation in the right lung were present when the patient was first seen with intestinal symptoms, but the consolidation was thought to be inflammatory in nature. Radiography after the second operation, however, demonstrated a large tumour deposit in the right lower lobe.

The appearance of the intestinal tumours found at the first laparotomy should, in retrospect, have made us alert to the possibility of their being secondary deposits. At this time, however, there was no clear clinical evidence of tumours elsewhere. The history of a simple mastectomy 4 years previously was thought at first to be irrelevant in view of the diagnosis of 'fibro-adenoma' that had been made. In fact, simple fibro-adenomas are not commonly seen in women in their sixties, and the original pathological report on the breast tumour stressed its large size and the large amount of stroma present; again, in retrospect, these are features which might have enabled us to reach a correct diagnosis earlier. This large fibro-adenoma is one which we would classify as a 'cystosarcoma phylloides'.

'Cystosarcoma phylloides' goes under many names. In the past, its giant size has been considered to be one of its most characteristic features, and its benign nature has been emphasized (McDonald and Harrington, 1950). Treves and Sunderland (1951) described 77 tumours which they classed as 'cystosarcoma phylloides'. They believed that the essential feature of these mixed epithelial and connective-tissue tumours of the breast was a comparative overgrowth of the stromal element; large size was not, in their view, an essential feature of the tumour. Of their 77 cases, 41 were "obviously benign", and 12 were "obviously malignant", that is, they were recurrent or metastasizing growths from which 8 patients had died. Twenty-four cases were at first classed as "doubtful", but after analysing the histological features in the malignant group, the authors reclassed 6 of the 24 as malignant. They emphasize, moreover, that more of the 'doubtful' group might later have to be classed as 'malignant', depending on their clinical behaviour. Lester and Stout (1954), applying Treves and Sunderland's criteria to their material, classed 20 out of their 58 cases of cystosarcoma phylloides as at least histologically malignant. The study of this group of breast tumours is made difficult by the inconsistent and varied terminology, but evidence is accumulating that the giant fibro-adenoma (cystosarcoma phylloides, or intracanalicular myxoma) has a greater capacity for behaving as a malignant tumour than is generally realized. It is certainly true that histological examination alone is at present not a reliable guide to the prognosis of these mammary tumours. The growth in our patient would, using Treves and Sunderland's criteria, fall in the 'doubtful' group; but four years later she was dead with widely disseminated metastases. The patient who has had a fibro-adenoma of this pattern removed, whether truly 'giant' or not, should, we believe, be most carefully followed-up; the behaviour of these tumours is not at present predictable with any confidence.

SUMMARY

A case is described of recurrent intussusception of the jejunum in a 66-year-old woman. Two operations were performed at an interval of three months, and in each case the intussusception was caused by a polypoid sarcomatous tumour arising in the submucosa of the jejunal wall. In each case, also, additional discrete tumours were found in the resected portion of gut.

Subsequent necropsy showed widely disseminated sarcomatous tumours in the right lung, œsophagus, liver, pancreas, adrenals, kidneys, thyroid, and brain.

Four years previously a breast had been removed for a 'fibro-adenoma'. Re-examination of this tumour revealed it to be a giant fibro-adenoma (cystosarcoma phylloides). It is believed that this tumour was the primary source of the metastatic sarcoma.

Secondary tumours of the intestine are a recognized cause of intussusception in adults.

The malignant potentialities of the 'cystosarcoma phylloides' of the breast, and the difficulty of assessing these on purely histological grounds, are stressed.

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THE PROBLEM OF SPONTANEOUS RUPTURE OF THE NORMAL ALIMENTARY CANAL

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RUPTURES of the alimentary tract occurring proximal to an obstruction or at the site of intrinsic disease, and those which result from trauma, are all well recognized in clinical practice. This paper is not concerned with these relatively common types of perforations, but discusses the problem of rupture in the much rarer group of cases in which a perforation takes place spontaneously in what appears to be a normal alimentary canal.

Six instances of such perforations are presented. Two occurred in the œsophagus, the third in the stomach, the fourth in the small bowel, and the last two in the colon.

THE ŒSOPHAGUS

Boerhaave (1724) recorded the first case of spontaneous rupture of the œsophagus, the patient being Baron de Wassenaer, the Lord Admiral of the Dutch Fleet. Ridgeway and Duncan (1937) found 36 cases in the literature, amongst which there were no survivors. In recent years excellent reviews of the condition have been written by Barrett (1946), Kinsella, Morse, and Hertzog (1948), and Mackler (1952). Increasing familiarity with the very striking clinical picture has led to more and more cases being diagnosed pre-operatively and successfully treated. Along with this greater clinical awareness, there has been a corresponding increase in the number of case reports, and Derbes and Mitchell (1956) found that the number of cases recorded had risen to 157, of which 55 had been treated surgically with 35 survivals.

Case 1.—R. N., a night watchman aged 77 years, was admitted to the General Infirmary at Leeds on Nov. 7, 1951. Three hours previously, while he was drinking beer in a public house, he suddenly felt nauseated but managed to suppress the impulse to vomit until he got outside. As soon as he started to vomit, he felt a sudden severe pain in his epigastrium which quickly increased in severity, spreading all over his abdomen and radiating behind the sternum into his left chest. Morphine ¼ gr. given by his own doctor did not relieve the pain.

On examination he was collapsed, sweating, and cyanotic, with short, grunting respiration. The severe pain in his chest and back prevented him from lying flat. His abdomen was rigid all over, but moved slightly on respiration. There was surgical emphysema at the root of the neck on both sides, but no abnormal signs in his chest. A clinical diagnosis was made of spontaneous rupture of the œsophagus.

Thoracotomy (Professor Allison) was performed seven hours after the onset of symptoms. The left ninth rib was excised and on opening the left pleural cavity about a pint of turbid fluid was found, but there was no direct communication between the œsophagus and the pleural cavity. The mediastinal pleura was greenish-black in colour and on incising it there was a whiff of gas and a large quantity of dark fluid welled up. The posterior mediastinum was then widely opened and the fluid sucked away. A linear tear, 1½ in. long, was found in the left anterior wall of the œsophagus which extended down to within a ½ in. of the diaphragm. The wall of the œsophagus appeared a little œdematous but was otherwise quite normal. The rent was repaired in two layers (mucosa and muscle coat) with catgut and the chest closed with an underwater drain. The patient made an uninterrupted recovery and was discharged home on Dec. 14. When seen four months later in the Out-patient Department he was free of all symptoms.

DISCUSSION

The prelude to a spontaneous rupture of the œsophagus is a rise in the abdominal pressure, usually due to a violent attack of vomiting, brought on by gluttony or an alcoholic spree. Cases have, however, been reported as occurring during defæcation (Whipple and St. John, 1941), an epileptic attack (Klein and Grossman, 1943), and childbirth (Kennard, 1950).

Best and Taylor (1950) describe the onset of vomiting as being initiated by the upper half of the stomach losing its tone and appearing as a "perfectly flaccid bag". This is followed by a series of contractions, starting at the middle of the stomach and ending in a contraction ring at the incisura, which divides off the pyloric portion of the stomach from the remainder. Next there is a powerful contraction of the diaphragm and the abdominal muscles, which ejects the stomach contents through the open cardia into the lower œsophagus. At the pharyngeal end the cricoid sphincter relaxes and at the same time the hyoid and larynx are drawn forward, so that the pharynx is enlarged and allows a free passage for the vomitus into the mouth.

An obstruction at any level of the œsophagus during vomiting will result in the pressure below the block rising precipitately and a danger of the wall tearing. Meyer (1858) had a patient, who was a cobbler, in whom an old stricture of the œsophagus became blocked by a piece of sausage and the attempts to dislodge it by vomiting led to rupture of the œsophagus below the site of the stricture. Fitz (1877) had a rather similar case in a sailor, in whom the obstruction was caused by an impacted lump of meat, and here again the vomiting culminated in a fatal rupture of the lower œsophagus. There is a general agreement that in a spontaneous rupture, although there is no organic obstruction, a functional one is provided by the failure of the cricoid sphincter to relax. The reason for this is unknown, but most authors seem to favour a theory of 'muscular incoordination'. The act of vomiting is an extremely complicated one, involving a series of co-ordinated reflexes, and it is reasonable to assume that repeated vomiting may lead to fatigue of the medullary centre so that the various muscle groups get 'out of step'. Mackler (1952) suggests that an aggravating cause may be the constant regurgitation of the hydrochloric acid, which produces a spasm of the sphincter. Another important factor may be that many of the patients, like the one reported in this paper, try to suppress the impulse to vomit by contracting the

cricopharyngeus and thus set the stage for a rupture of the œsophagus at a lower level.

Since the condition was first clinically recognized, there have been numerous experiments performed (McKenzie, 1884; Kinsella and others, 1948; Mackler, 1952; Bodi, Fanger, and Forsythe, 1954; Derbes and Mitchell, 1956) in which the œsophagi of corpses have been ruptured by forcing air into them under pressure. In almost every instance the result was a longitudinal tear in the lower third of the œsophagus, which was identical to those occurring in spontaneous ruptures. Most authors agree with Mackler that an average pressure of 5 lb./sq. in. is sufficient to rupture an œsophagus, and Bodi and others produced tears with a pressure as low as 3·5 lb./sq. in. It is not, however, so much a question of the absolute level of pressure as the speed with which it is built up. Burt (1931), in experiments at all levels of the alimentary tract, found that a relatively small pressure applied quickly could produce a rupture just as effectively as a much larger pressure applied slowly. Unfortunately no exact information is available about the pressures in the œsophagus during vomiting. Kinsella and others state that in forced expiration healthy adults reach pressures of 2·1 lb./sq. in. and even in some cases can go to 3·4 lb./sq. in. This last figure is dangerously near the minimal pressure which produced tears in the œsophagus and there seems no good reason to doubt that in an obstructed œsophagus during vomiting the pressures can rise considerably above these levels.

The reason for the ruptures almost always being longitudinal and linear was investigated by Derbes and Mitchell (1956). After a long and involved mathematical analysis of the hydrodynamic forces involved, they concluded that "the mechanical factors incident to increased internal pressures compel the œsophagus to tear in a vertical plane".

There remains the question of why the rupture occurs in the lower third of the œsophagus. Bodi and others (1954) believe that there is an inherent weakness at this site owing to the entrance of vessels and nerves into the musculature, and that this is made worse by segmental defects in the circular muscle coat. Mackler (1952) puts forward three reasons to account for the lower part tearing: First, smooth muscle entirely replaces striated muscle; secondly, that whereas the trachea strengthens the upper half of the œsophagus, the lower part is unsupported; and thirdly, that the longitudinal muscle-fibres tend to splay out in the lower third as they descend to join the stomach and no longer form a continuous encircling coat.

Case 2.—J. T., a 14-year-old schoolboy, was admitted to the Radcliffe Infirmary on Jan. 1, 1957. Fourteen days previously he had sustained a severe gunshot wound of the right side of his head, with a considerable loss of bone and soft tissues. He was admitted to the Leicester Royal Infirmary where the wound was closed by mobilizing skin-flaps, but it unfortunately broke down and began leaking cerebrospinal fluid.

On examination he was conscious but had signs of extensive damage to the right cerebral hemisphere with an aphasia, a left homonymous hemianopia, and a left hemiplegia. There was an open wound in the right temporal and zygomatic regions which was leaking cerebrospinal fluid and through which a cerebral fungus, 3 cm. in diameter, was protruding. A lumbar puncture confirmed the presence of a fulminating meningitis due to Gram-positive and Gram-negative cocci.

Further lumbar punctures were unsuccessful and on Jan. 2 bifrontal burr holes were made and intraventricular penicillin and streptomycin started. Five hours after this operation he suddenly collapsed and died within a matter of minutes.

Post-mortem examination showed a widespread meningitis with extensive damage to the right side of the brain. The pleural cavities were full of brown fluid and there was a hole 4 cm. in diameter in the anterior wall of the œsophagus just above the diaphragm. There was also an acute erosion in the fundus of the stomach. The pathologist thought both changes preceded death.

Cushing (1932), in a review of 11 cases, drew attention to the curious relationship between gastric and duodenal ulceration and intracranial lesions. He thought that the ulceration was due to a hypothalmic disturbance which caused overstimulation of the vagi. Amongst the cases he reported was that of a 10-year-old boy who died following incomplete removal of a cerebellar medulloblastoma and who was found post mortem to have a ruptured œsophagus. Fincher and Swanson (1949) reported 3 cases in which benign intracranial tumours had been removed and which died in the immediate post-operative period of ruptured œsophagi. They seem to have accepted Cushing's theory that the lesions were probably due to ulceration of the lower end of the œsophagus. Priveteri and Gay (1951) recorded 4 fatal cases of intracranial lesions, in all of which a ruptured œsophagus was found post mortem. They make no comment, however, on a possible connexion between the two conditions. McIver, Smith, Tomlinson, and Whitby (1956), in 452 autopsies on patients with intracranial disease, found 15 with ruptures of the œsophagus. They point out that the tears were identical to those occurring in spontaneous ruptures and suggest that post-operative vomiting and retching were their cause. In none of their cases was there convincing evidence of œsophageal ulceration, but they admit that a hypothalmic disturbance may be partly responsible by causing muscular incoordination during vomiting.

I have examined 300 consecutive post-mortem reports on cases admitted to the neurosurgical unit at the Radcliffe Infirmary. In 100 instances death was due to a head injury and in the remaining 200 there was an intracranial lesion. In only 2 patients was a ruptured œsophagus found. In the first case death followed a head injury and the perforation (which was just above the diaphragm) was considered by the pathologist to be the result of post-mortem digestion. The second patient was a spastic idiot who died in coma, and the perforation (which was described as 'a split') was found in the anterior œsophageal wall opposite to the tracheal bifurcation. In this second case the pathologist was uncertain whether the rent occurred before or after death; the histology suggested it was an ante-mortem event.

It is difficult to reconcile these figures with those of McIver and others (1956), but they hardly support these writers in their view "that a relationship exists between certain lesions of the central nervous system and a rupture of the œsophagus".

Clinical Picture.—The symptoms and signs of spontaneous rupture of the œsophagus have been

fully described by Barrett (1946) and Anderson (1952) in their reviews of the subject. Most cases occur in middle-aged, overweight men (women are rarely affected), and the symptoms almost always come on during an attack of vomiting following a large meal or a bout of drinking. Shock, pain, and respiratory distress are the initial symptoms. The pain may be epigastric or substernal, and frequently radiates to the back. It is exceptionally severe and many writers comment that morphine fails to relieve it. Severe thirst is a symptom frequently mentioned in the case histories. Surgical emphysema at the root of the neck is a valuable sign, but unfortunately is only present in 60 per cent of cases. Clinically, the common mistake is for the surgeon to diagnose a perforated peptic ulcer and the physician a coronary thrombosis. Both are serious errors, leading in the one case to a useless laparotomy and in the other to a fatal delay in treatment.

X-ray examination of the chest may aid the diagnosis by showing mediastinal emphysema in the early stages and a hydropneumothorax later. It may be possible to demonstrate the presence of a fistula by making the patient swallow lipiodol and then taking a radiograph of the chest. Another method of achieving the same end is to give a dye by mouth and then aspirate the free fluid in the pleural cavity to see if it has been stained.

Treatment.—Barrett (1947) reported the first successful repair of a spontaneous perforation of the œsophagus. There is a general agreement that in the first few hours immediate thoracotomy and repair of the tear holds out the best chance of survival for these cases. Samson (1951), while strongly advocating this approach, suggests that there may be a case for simple drainage of the pleural cavity in those very rare patients who survive forty-eight hours before being seen. He bases this view on the fact that by then infection of the œsophagus, mediastinum, and pleura will be well established and an attempt to close the tear is likely to fail. Even so, he points out that cases so treated have a long and complicated convalescence.

THE STOMACH

Perforation of a gastric ulcer is an everyday occurrence but rupture of the stomach itself is fortunately very rare, because it is almost invariably fatal. Camerarius (1714) described a case in a young girl, who, after eating a large number of green damson plums, was seized by severe abdominal pain and died within a few hours. The post-mortem examination showed a tear in the fundus of the stomach. Woolf (1936), reviewing 17 cases of spontaneous rupture of the stomach, concluded that overdistension was the cause in the majority of cases. Leger and Maes (1947) found 30 cases in the literature and agreed with Woolf that overdistension was the chief reason for the rupture. Ladkin and Davies (1948) reported a case in a 5-year-old African boy who had eaten a large number of sweet potatoes. They thought the rupture was probably due to "excessive fermentation". Burket (1949) described a case in a nun who had taken an excessive amount of sodium bicarbonate to relieve her indigestion. Lemmon and Paschal (1941) had a similar case. Rejithar's (1952) patient had a pyloric stenosis and had been vomiting for some weeks before finally rupturing her stomach. Millar, Bruce, and Patterson (1957) described 2 cases; the first had a long-standing gastric ulcer with pyloric stenosis; the second had "discomfort in the upper abdomen for which she had been taking baking soda".

Case Report.—E. C., a pipe leader, aged 61, was admitted to the Radcliffe Infirmary on July 12, 1959. Five hours previously, following a substantial lunch, he had sudden severe pain in his large left inguinal hernia which rapidly became even bigger in size. There was no history of trauma. On examination he was in obvious pain and, although his blood-pressure was only 90/60, his general condition was good. The left side of the scrotum was filled by an enormous inguinal hernia which stretched down to the patient's knees. It was very tender and the contents were irreducible. There was guarding all over the abdomen, although this was not very marked. A clinical diagnosis of a strangulated left inguinal hernia was made. At operation (K. C.) the hernial sac was found to be filled with nine pints of black fluid in which floated pieces of undigested food. No perforation could be found in the bowel contained in the sac (about half of the small bowel and nearly all the colon), so the peritoneal cavity was opened through a lower left paramedian incision.

A linear tear, 6 cm. long and involving all the coats of the stomach, was found half-way along the lesser curve. Apart from the fact that the stomach seemed large there was no other abnormality. The tear was sutured in two layers with thread, the peritoneum sucked out, and the wounds closed. No attempt was made to repair the hernia. In spite of intensive efforts to maintain his blood-pressure, the patient's condition steadily deteriorated and he died forty-eight hours after the operation.

The autopsy revealed that there were two further incomplete mucosal tears in the region of the lesser curve. The histological sections taken from these mucosal rents showed acute ulceration which extended down to and involved the muscularis mucosæ while sparing the muscle coat itself. At the site of the complete rupture there was necrosis of the gastric wall and acute inflammation. There was no fibrous or obliterative endarteritis in any of the sections examined. Sections taken from the remainder of the stomach showed evidence of chronic gastritis.

DISCUSSION

The cause of the rupture is probably much the same as in the œsophagus—namely, sudden overdistension of a hollow viscus which is closed at both ends. The overdistension is usually due to overeating or over-drinking and some patients have made it worse by taking sodium bicarbonate to relieve the resulting indigestion. Millar and others (1957) suggest that swallowed air and possible gaseous transfer from engorged vessels may still further raise the intragastric pressure. Many cases have an organic stenosis at the pylorus from an ulcer or a growth; in others one must assume that there is a functional obstruction due to pylorospasm. The failure of the cardiac sphincter to relax in the face of a rapidly mounting pressure is not fully understood. It may be that the overdistension (which may be largely gaseous) comes on so suddenly that there is no time for the cardia to open and thus lower the intragastric pressure.

Most ruptures are linear rents along the lesser curve. Frankel (1906) says that this is the least distensible part of the stomach and thus the most likely to tear. Key-Aberg (1891) believes that the stomach forms a sphere when distended, so that the relatively rigid lesser curve becomes overstretched and ruptures.

Clinical Picture.—Millar and others (1957) say that the following tetrad should suggest the diagnosis: (1) Tympanitic abdominal distension; (2) Rigidity of the abdominal parietes; (3) Subcutaneous emphysema; (4) Shock.

Treatment.—Unlike perforation of a peptic ulcer, rupture of the stomach is almost invariably fatal. Twenty-four of Leger and Maes's (1947) series of 30 patients died before any attempt could be made to explore the abdomen, and of the 6 patients who were operated on and the rent closed there was only 1 survivor. The reason for this appalling mortality when compared with that of a perforated peptic ulcer is not clear, but it may be a matter of relative sizes. A perforated peptic ulcer is rarely bigger than $\frac{1}{2}$ in., whereas most ruptures are about 2 in. long and some have extended for 5 in. along the lesser curve. This must mean that there is more peritoneal contamination in a rupture with a correspondingly greater degree of shock. Certainly on reading the case reports, one noted how gravely ill these patients were, most being almost *in extremis* from the start.

THE BOWEL IN NEONATES

Two different types of peritonitis are found in the newborn—namely, meconium and acute bacterial (Rickham, 1955). The first is due to the escape of meconium into the peritoneal cavity, and Boikan (1930) says that the term "should be applied to cases resulting from fœtal or intrapartum or shortly postpartum intestinal rupture". The peritonitis, thus established, is chemical and sterile and is characterized by widespread adhesion formation, so that many of these infants present with intestinal obstruction.

At birth the bowel is sterile, but after twenty-four hours it is invaded by pathogenic bacteria so that any perforation occurring after this time will result in an acute bacterial peritonitis. (An originally sterile meconium peritonitis can, of course, become secondarily infected and thus converted into a bacterial one.)

Boikan has claimed that spontaneous perforation of the bowel accounts for half the cases of meconium peritonitis, the remainder being due to intestinal obstruction. Forshall, Hall, and Rickham (1952) disagree with this statement and maintain that many cases so described were, in fact, examples of acute bacterial infection. They believe that the majority of cases of meconium peritonitis are due to an organic intestinal obstruction and only a small minority to spontaneous perforation.

Leaving aside this controversy about nomenclature, there is no doubt that spontaneous perforations of the bowel can and do occur both *in utero* and in the newborn infant. Thelander (1939) collected 39 examples from the literature, and further cases have been added by Agerty, Ziserman, and Shollenberger (1943), Lattes (1943), Franklin and Hosford (1952), and Standard (1952).

Case Report.—R. G., a male infant five days old, was admitted to the Radcliffe Infirmary on May 24, 1957. The pregnancy and labour had been uncomplicated and the baby appeared normal at birth. Eight hours after delivery he vomited bile-stained fluid for nine hours. Over the next three days he would only take small amounts of breast milk and two days before admission again began vomiting bile. On examination the temperature was 98°, pulse-rate 150, and respirations 32. In spite of the vomiting he looked well and was not obviously dehydrated. There was gross abdominal distension and a plain radiograph of the abdomen showed distended coils of small intestine. At laparotomy (Mr. Elliot-Smith) there was turbid straw-coloured free fluid in the abdomen. One loop of small bowel was congested and contained areas of ecchymoses, one of which had perforated. The colon was distended but there was no evidence of any atresia in the bowel. The perforation was closed by simple suture. After an initial satisfactory recovery the infant collapsed and died on the fourth post-operative day. Post-mortem examination revealed a general peritonitis with a second perforation in the originally affected loop of bowel. This was situated 60 cm. proximal to the ileocæcal valve. There was no abnormality in the remainder of the bowel, and histological examination of the perforated segment showed no evidence of any muscular defects. Cultures of the bowel contents grew *Bacterium coli* and *Streptococcus fæcalis*.

DISCUSSION

In the cases without an intestinal obstruction, the reason for the spontaneous perforation of apparently normal bowel has long been a subject of debate. Various theories have been suggested, but it is probably true to say that no one explanation is sufficient to account for all cases.

1. Congenital Defects in the Bowel Wall.—Weakness or defects in the muscular coat of the bowel may cause a certain number of spontaneous perforations. Moretti (1949) has reported a case of meconium peritonitis in association with a distal atresia. He found that there were wide gaps in the circular muscle coat which he considered were of congenital origin and that the perforation had occurred through one of these. Davis and Poynter (1922) have reviewed 401 cases of intestinal obstruction in infancy without finding a single example of the bowel perforating. This lends support to the view that even in obstructed cases there must be a congenital weakness of the gut before it will rupture. Herbut (1943) and Pendergrass and Booth (1948) have recorded ruptures of the stomach in the newborn due to defects in the muscle coat and it is reasonable to assume that perforation of similar weakened areas in the bowel can take place even in the absence of a distal obstruction.

Boikan (1930) and Lattes (1943) thought that the perforations in their cases were due to hypertrophy of the intestinal glands. The histology of the perforated segments of bowel showed submucosal lymphoid hyperplasia causing atrophy of the overlying muscle coat. This was accompanied by enlargement of the glands of Lieberkühn, which penetrated the disorganized musculature and finally ruptured through the serosa. These two cases, however, seem quite unique and in none of the other reports is there any mention of abnormalities in the intestinal glands.

2. Trauma during Delivery.—Zillner (1884) suggested that trauma during labour was the most likely cause of the perforation. In his paper he reported 4 infants who died within the first few days of life and who were found at autopsy to have tears in their sigmoid colons. He thought that the compression exerted by the uterine contractions was responsible for the injuries. His views are, however, somewhat invalidated by the fact that all four infants had been given enemata by inexperienced midwives.

It is more likely that it was the traumata of these enemata, rather than the uterine contractions, which were responsible for the perforations.

Moreover, one has to remember that the bowel is empty of gas before birth and no compression force, whether due to the contracting uterus or the hand of an obstetrician, is likely to rupture loops of gut lying collapsed on the posterior abdominal wall.

Although Russell (1928) supports Zillner in his views, no other writer is prepared to accept trauma during delivery as a cause of the perforation.

3. Infection of the Bowel.—Abt (1931) believed that the perforations were due to infection of the bowel transmitted from the mother to the fœtus. Thelander (1939) in her discussion also felt that the role of infection had been underrated in the past. She points out that the sterility of the fœtus *in utero* is a very relative matter and claims that there is a direct correlation between the length of labour and the frequency of positive cultures of placental and cord blood. Prolonged labour also leads to fœtal asphyxiation which, according to Guthrie (1942), causes mucosal hæmorrhages in the gastro-intestinal tract. Such an area of mucosal necrosis may become infected either from the blood-stream or by the pathogens which appear in the bowel after twenty-four hours. Under these circumstances a rapidly perforating ulcer may be readily produced.

In the case reported in this paper the sudden death of the infant was undoubtedly due to the bowel perforating for a second time. The congested state of the affected loop with patches of ecchymoses suggests an infective origin for the perforation. Moreover, the onset of systems, within a few hours of birth, rather favour an initial blood-borne infection because at this time the gut itself is sterile.

Clinical Picture.—If the bowel perforates while the infant is still *in utero*, the resulting abdominal distension may cause an obstructed labour (Lattes, 1943; Franklin and Hosford, 1952). More commonly an increasing abdominal distension appears after birth and this interferes with breathing so that the infant is cyanotic, with short, grunting respiration. Bilious vomiting and refusal to take feeds soon lead to a severe degree of dehydration.

A straight radiograph of the abdomen will show loops of dilated gut with a pneumoperitoneum which may be general or loculated. In cases of meconium peritonitis calcification may be seen and is diagnostic of the condition. Some authors (Franklin and Hosford) have suggested aspirating free fluid from the peritoneal cavity to see if it is stained with meconium. There is, however, the danger of piercing the bowel and, as Bentley and Waterson (1954) point out, there may in fact be no free fluid present.

Treatment.—As soon as the diagnosis is made, urgent laparotomy is required. In cases of meconium peritonitis it may be impossible to find any perforation and the bowel may be so matted together that the only feasible course is anastomosis of collapsed and dilated loops. If a perforation is found it should be closed by simple suture.

There were no survivors among Thelander's series of 39 cases, but White (1956) found 18 reports in the literature of successful surgical intervention in cases of meconium peritonitis.

Table I.—Adult Cases of Perforation of the Bowel

	Author	Sex	Age	Hernia	Precipitating Factor	Site of Perforation	Treatment	Outcome
1	Lespiau (1867)	M.	31 yr.	?	Defæcation	Ileum—70 cm. above ileocæcal junction	No operation	Death
2	Mikulicz (1885)	M.	40 yr.	R.I.H.	Getting out of bed	Ileum	Margins of perforation excised and bowel sutured	Recovery
3	Huntley (1919)	M.	34 yr.	?	Defæcation	Rectosigmoid junction	Suture of perforation	Death
4	Taylor (1925)	M.	66 yr.	L.I.H.	Straining to pass water	"Small intestine, about middle"	Suture of perforation	Recovery
5	Hahn (1931)	M.	37 yr.	B.I.H.	Defæcation	Sigmoid colon	Perforation sutured to abdominal wall to form temporary fæcal fistula	Recovery
6	Brown (1944)	M.	30 yr.	?	Nil	Sigmoid colon	Suture of perforation	Recovery
7	Lataix (1949)	F.	43 yr.	Incisional	Nil	Rectosigmoid junction	Suture of perforation and left iliac colostomy	Recovery
8	Weinstein and Roberts (1952)	M.	63 yr.	No	Defæcation	Sigmoid colon	Suture of perforation and cæcostomy	Recovery
9	Eadie (1955)	F.	57 yr.	No	Defæcation	Sigmoid colon	Perforated loop exteriorized	Recovery
10	Henderson (1957)	F.	38 yr.	No	Nil	Sigmoid colon	Perforated loop exteriorized	Recovery
11	Henderson (1957)	F.	49 yr.	No	Nil	Rectosigmoid junction	Perforation closed and proximal colostomy made	Recovery
12	Cronin (1959)	M.	41 yr.	L.I.H.	Sneeze	Sigmoid colon	Suture of perforation and transverse colostomy	Recovery
13	Cronin (1959)	F.	69 yr.	No	Nil	Sigmoid colon	Perforated loop exteriorized	Death

THE BOWEL IN ADULTS

Spontaneous perforation of the normal bowel in the adult is very rare. It may occur during defæcation, micturition, or, in some instances, without any obvious cause at all. A review of the literature has shown 11 such cases and the present 2 cases brings the total to 13. These 13 case reports are summarized in *Table I*.

Case 1.—E. B., a male school teacher aged 41 years, was admitted to the Radcliffe Infirmary on June 13, 1957. Five hours previously, while shaving, he had sneezed and immediately felt a severe pain in the hypogastrium which rapidly spread all over his abdomen. For twelve years he had had a reducible left inguinal hernia which had never given him any trouble. On examination he was an obese, ill-looking man; temperature 98·2 ; pulse 92; B.P. 120/80. His abdomen was rigid and acutely tender. Because of a rather vague history of pain after meals, a provisional diagnosis of perforated peptic ulcer was made. At laparotomy (K. C.), the peritoneal cavity was full of brown, fæculent smelling free fluid, but the stomach and duodenum were normal. The original incision was extended downwards and a linear tear 1½ in. long was found in the lower third of the sigmoid colon. It ran in the long axis of the bowel and lay midway between the mesenteric and antimesenteric borders. It had clean-cut edges through which the mucosa pouted. The remainder of the colon appeared normal, nor was it unduly full of fæces. The rent was closed and a proximal transverse colostomy made. The post-operative course was complicated by a severe wound infection and an abscess at the site of the colostomy. An acute renal failure also developed, possibly resulting from a prolonged period of hypotension after the operation. By the end of six months the patient's renal function had returned to normal and the colostomy was closed. Two barium enemata and a sigmoidoscopy have shown no abnormality in the large bowel.

Case 2.—G. R., a housewife, aged 69 years, was admitted to the Radcliffe Infirmary on May 25, 1959. For many years she had been taking alkalis for indigestion and she came into hospital with a history of seven hours' severe central abdominal pain. She had an obvious general peritonitis which was thought to be due to a perforated peptic ulcer. At operation (K. C.) the peritoneum was filled with fæculent smelling free fluid and there were lumps of fæces lying free in the pelvis. A linear tear, 4 cm. long, was found at the apex of the sigmoid colon. It lay on the antimesenteric border at right angles to the long axis of the bowel and had clean-cut edges. The colon above the rupture was filled with hard fæces but was otherwise quite normal. The perforated loop was exteriorized as a left iliac colostomy and the abdomen closed. The patient died on the following day. The histological sections through the site of rupture showed no abnormality in the bowel.

DISCUSSION

Site and Cause of Perforation.—In this series of 13 cases of spontaneous rupture of the bowel, 3 ruptures involved the small bowel and 10 the large. All 3 cases of small-bowel perforation occurred in men; 2 had inguinal herniæ and in 1 the presence or otherwise of a hernia is not mentioned. In 2, the site of perforation was in the terminal ileum and in 1 in "the small intestine, about the middle". Perforation occurred in 1 patient during defæcation, in 1 while straining to pass water, and in 1 while he was getting out of bed in the morning.

Of the 10 cases of ruptured large bowel, 5 occurred in men and 5 in women. Inguinal herniæ were present in 2 patients and an incisional hernia in 1, but in 5 it is definitely stated that no hernia was present. No mention is made of a hernia in the remaining 2 patients. The sigmoid colon was the segment of bowel affected in all 10 cases. In 4 patients perforation took place during defæcation and in 1 it followed a sneeze. In 5 cases there was no obvious cause of the perforation.

Classification.—The 13 patients in this series fall into three groups, depending on the presence or not of an inguinal hernia and whether the abdominal pressure was raised or not at the time of the intestinal rupture.

Group I. Patients who had both an inguinal hernia and a raised abdominal pressure.—Four of the patients (Mikulicz, 1885; Taylor, 1925; Hahn, 1931; present author) had inguinal herniæ and the perforation in each case was preceded by a rise in the abdominal pressure; 2 occurred during defæcation, 1 while getting out of bed, and 1 after a sneeze.

Wilenski and Kaufman (1937) pointed out the association between rupture of the bowel, muscular effort, and the presence of a hernia. The essential point they made was that in a patient with a hernia a sudden rise in the abdominal pressure may lead to perforation of a loop of gut.

There are three possible explanations of why this may happen:—

1. Bunge (1905) suggested that normally a rise in the intra-abdominal pressure is instantaneously followed by a raised intra-intestinal pressure, and the two being equal, perforation does not occur. If, however, the bowel is lying against (not in) the neck of a hernial sac the sudden rise in pressure would force it against the sac, so sealing the latter off from the general peritoneal cavity. The pressure in the sac would in consequence be lower than in the gut, resulting in rupture of the latter. The perforated loop then falls back and results in a general peritonitis.

2. Haim (1910), on the other hand, believes that the bowel and abdominal wall should be considered as a double-walled tube around a single cavity, i.e., the lumen of the gut. If a rise in the abdominal pressure occurs, any weakness in the outer wall (e.g., a hernia) will lead to rupture of the inner wall (the gut).

Both of these theories assume that the gut 'bursts' as the result of a sudden rise in pressure so that the perforation is comparable to that seen in compressed-air injuries of the colon.

3. Another theory is put forward by Aird (1937) in reporting 2 cases similar to those of Wilenksi and Kaufman (1937). He says, "it would be simpler to explain these cases by overstretching of a loop of bowel fixed at one end at the ileocæcal valve or duodeno-jejunal junction and gripped at the other by contraction of the abdominal muscles around the hernial orifice".

Of the 4 patients, 1 (Mikulicz) had a right inguinal hernia with the perforation in the terminal ileum. In this patient Aird's theory of overstretching of the bowel is probably correct. It is less easy to explain the remaining 3 cases on the same view. In 2 of them (Hahn, 1931; present author) the sigmoid colon was perforated. Now while it is rare to find the pelvic colon lying free in an inguinal hernia, it is common for it to be placed just outside the neck of the sac, and it is precisely under these conditions that a sudden rise in the abdominal pressure may lead to

sudden rupture of the bowel. Similarly, it seems more likely that in the third patient (Taylor, 1925) the perforation in "the small intestine, about the middle" was due to a 'burst injury', because it is difficult to see how such a loop could be overstretched.

Group II. Patients who had no definite inguinal hernia but did have a raised abdominal pressure.—In four patients (Lespiau, 1867; Huntley, 1919; Weinstein and Roberts, 1952; Eadie 1955) the perforation occurred while the patient was defæcating and it was presumably this strain that led to the bowel tearing. Weinstein and Roberts and Eadie state definitely that no hernia was present in their patients, but the other two authors do not say whether one was present or not.

Robinson (1938) records a case of spontaneous perforation of the small bowel which occurred while the patient was racing between bases in a ball game. The interest of this case lies in the fact that although no hernia was found either on admission (the internist noted "both inguinal rings small") or at any time on subsequent examination, the patient when closely questioned admitted to having had an intermittent swelling in the left groin for many years. Moreover, he said that he felt that he had been "ruptured" when the pain first came on. Robinson concluded that a small hernial sac was almost certainly present at the time of the perforation.

The significance of this case to the 4 patients in this second group is obvious. It emphasizes the fact that although no hernia may have been found on examination, this does not exclude there having been one present when the bowel ruptured.

Eadie has suggested another possible explanation for the perforations in the sigmoid colon. He points out that in the early stages of diverticulosis, although the gut wall appears normal, the muscle coat may be extremely attenuated and is thus more liable to rupture than the normal bowel. Using freshly excised colon, Burt (1931) has shown that a pressure as small as 3 4 lb. per sq. in. is sufficient to produce a perforation. Presumably, a still smaller force might be sufficient to rupture a colon weakened by diverticulosis. Recently a patient (Corry, 1958) was admitted to the Radcliffe Infirmary with an apparent spontaneous perforation of the colon. At operation the tear was found at the rectosigmoid junction and the bowel wall appeared quite normal. Subsequent investigation showed a few isolated diverticula in the descending colon. Probably the perforation in this case occurred either through a solitary diverticulum (there was, however, no evidence of this at operation) or else, as Eadie suggests, in a portion of the wall weakened by the muscular atrophy of diverticulosis.

Group III. Patients who had neither an inguinal hernia nor a raised abdominal pressure.—It is more difficult to find a satisfactory explanation for the perforation of the bowel in the last 5 patients (Brown, 1944; Lataix, 1949; Henderson, 1957—2 cases; Cronin, present paper).

Brown makes no mention of a hernia in his patient, but in any case there was no rise in the abdominal pressure before the onset of symptoms. The possibility of a diverticulosis is ruled out because a post-operative barium enema showed no abnormality in the colon. There was no sign of any foreign body in the bowel and the patient vigorously denied rectal interference. Brown concludes his case report by saying, "I can offer no explanation of the cause of this condition".

Lataix's patient was a peasant woman who had a large incisional hernia through an old appendicectomy scar. The perforation at the rectosigmoid junction was very small and neither the history nor the operative findings gives any clue as to its possible cause. Admittedly, this patient did have a hernia, but it is difficult to believe that it could be in any way responsible for the perforation.

Henderson's first patient had been complaining of abdominal pain for fourteen days before admission and she was in hospital for two days before being explored. A circular perforation of the sigmoid colon was found at operation and "the lumen of the gut was filled with firm, but not hard, fæcal material which was blocking the perforation". The affected segment of bowel was removed, but nothing unusual was found in its histology. The second patient suffered from chronic constipation and, in contrast to the first, the pain came on suddenly and she had an obvious clinical peritonitis. In this patient, "very hard fæcal masses were present in the left colon, and near the rectosigmoid junction there was a vertical linear tear in the bowel". The present author's *Case* 2 had almost the same clinical and operative findings as those of Henderson's second patient. It is tempting to dismiss these three cases as examples of perforated stercoral ulcers. Barron (1928), however, reviewed 53 such cases, and in the majority of them found that the edges of the ulcer were thickened and indurated. Henderson, on the other hand, referring to his first patient, specifically states, "there was astonishingly little inflammatory reaction even at the margin of the defect". He, like Brown and Lataix, was at a loss to explain the operative findings in his two patients. One cannot, however, help but feel (particularly in the second case) that the large fæcal masses in the colon were in some way responsible.

There are instances of what appears to be an unexplained spontaneous perforation, the cause of which comes to light in the course of time. Thus, Woodruff (1952), in a review of 14 cases of perforation of the colon, quotes one patient in whom the rupture occurred in the upper third of the rectum which otherwise appeared quite normal. Three months later a large carcinoma was obvious at the site of the perforation and was almost certainly its original cause.

There remain, however, a small number of patients illustrated by those in this last group in whom the perforation is not only 'spontaneous' but at present inexplicable.

Clinical Picture.—Except for one (Henderson's first case) all these patients presented with a history of severe pain of sudden onset and on examination had the signs of a general peritonitis. It is interesting that in *Case* 1 reported in this paper there was quite marked tenderness over the empty hernial sac, presumably because it was involved in the general peritonitis. In no instance was a correct pre-operative diagnosis made.

Treatment.—Simple suture of the perforation, with a proximal colostomy in the case of the large bowel, is the treatment favoured by most writers.

There were only 3 deaths (Lespiau, Huntley; Cronin, present paper) in this series of 13 patients and Lespiau's patient was not operated on. Considering that in 8 of the remaining 10 patients the perforation was found in the sigmoid colon, this is a surprisingly low mortality.

CONCLUSION AND SUMMARY

The term 'spontaneous' when applied to ruptures of the alimentary tract has often been thought to be synonymous with 'idiopathic'. The study of the cases described in this paper, however, lead one to conclude that in most instances a rational and adequate explanation can be given for a perforation of an apparently normal viscus.

The causes of such perforations can be summarized as follows:—

1. A sudden rise in the intraluminal pressure in a hollow viscus, closed at both ends. It is under such conditions that the œsophagus and stomach rupture.

2. A sudden rise in the intraluminal pressure in the bowel without a corresponding rise in the pressure outside one segment of that bowel. Such a set of circumstances occurs in a patient with an inguinal hernia who suddenly contracts the abdominal muscles. The detailed mechanism is discussed in this paper.

3. Defects in the muscular coat of the bowel, congenital, in infants, and acquired, as in adults with diverticulosis, may so weaken the bowel that it ruptures under minimal strain. There is an inherent weakness in the lower third of the œsophagus which probably accounts for its being the commonest site of rupture.

4. Infective ulceration of the bowel is probably the cause of many of the perforations found in neonates.

5. There is a small group of cases of spontaneous rupture of the intestine in adults for which so far there is no satisfactory explanation.

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RETICULOSARCOMA OF BOTH BREASTS

By PAUL STRINGER, St. Albans

Sarcomata form only 3 per cent of all malignant tumours of the breast. Of the sarcomata only a fraction appears to be derived from either lymphoid or reticulo-connective tissue, the majority arising from intracanalicular fibro-adenoma or related tissue malformation. The occurrence of reticulosarcomata in both breasts therefore appeared worthy of record, more especially in view of the interval before the second breast was affected and because of the close clinical resemblance to carcinoma.

CASE REPORT

The patient, a 53-year-old married woman, a primipara, first attended on Sept. 30, 1954, complaining of a small swelling in the upper and outer quadrant of the right breast. This had been present for seven months. During that period it had not noticeably increased in size. There had been no discharge from the nipple and no pain within the lump or the breast. Her general health was good.

Six years prior to her attendance she had had a left radical mastectomy carried out for a swelling in the left breast which had appeared to her to be similar to that of her present complaint. There were no other significant features in her past history.

On examination there was a small hard lump half an inch in diameter in the upper and outer quadrant of the right breast. The surrounding tissue showed a coarse irregular thickening. The whole affected area, apparently approximating 2 in. in diameter, was not lobar but of irregular contour and outline. There was a soundly healed left radical mastectomy scar. No enlarged glands were palpable in either axillæ or in the neck. Chest X-ray and blood-count were normal. The W. R. was negative.

The patient was admitted for local excision of the lump on Oct. 13, 1954. Histological report was as follows: Lobular hyperplasia of breast with minimal cystic change. The two most striking features of these sections are sclerosis of mammary connective tissue and the very intense and widespread collections of lymphocytes and mononuclears in connective tissue and fat. There is no evidence of malignancy.

Fig. 51.—Appearance of the right breast, showing the similarity to carcinoma.

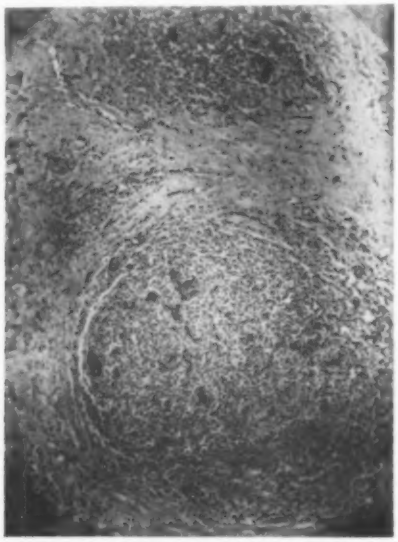

Fig. 52.—Section from right breast showing diffuse infiltration of breast tissue with lymphoreticular cells. (× 52.)

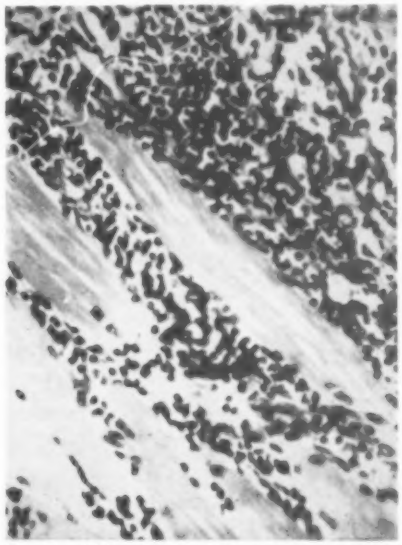

Fig. 53.—High-power view demonstrating lymphocytes and lymphoblasts as well as dictyocytic and syncytial reticulum cells. (× 280.)

The report concerning the previously removed left breast was now made available and was as follows.

Pathological Report on Left Radical Mastectomy, March 2, 1948.—Left breast with a tumour of indefinite outline above and behind the nipple which is neither retracted nor fixed to the mass. No fixation to the pectoral fascia. The tumour is a mass of 'chronic mastitis' heavily

infiltrated by lymphocytes and other mononuclears of varying size and shape. The epithelial hyperplasia—adenosis—is disrupted and overrun by this infiltrate which, with bands of dense fibrosis, is responsible for the induration.

It was considered that the histological appearance of the section from the right breast was identical with that from the left.

The patient attended six months later, when a fairly hard nodule at the site of the local excision in the right breast was noted. This was kept under observation. A year later no obvious change in the nodule was evident, but further follow-up in May, 1957, showed the presence of an irregular hard mass with ill-defined outlines infiltrating both skin and muscle in the left half of the right breast, with œdema of the nipple and areola (*Fig.* 51). The appearances strongly suggested carcinoma of the breast. The absence of enlarged glands on clinical examination in both axillæ and in the neck was confirmed, and the possibility of sarcomatous change was considered. A simple mastectomy was performed and the underlying muscle excised where infiltration appeared to have recurred. Skin-grafting was necessary, but healing was uneventful. The sections were then reported on as follows.

MACROSCOPIC.—Breast disk completely covered by an ellipse of skin 13 × 20 cm. on the front, and some striped muscle-fibres and fat on the deep surface. The nipple is large and not retracted. The skin of the inner hemisphere, particularly the upper quadrant, is raised and lobulated with a patchy cyanotic flush of the skin. It extends from the areola to within 1 cm. of the edge. The whole breast substance is replaced by fleshy glistening grey substance intersected by white mesh-work with abundant fat at the periphery.

MICROSCOPIC.—The breast is enlarged from diffuse infiltration of lymphoreticular cells which replace much of the breast tissue proper. However, isolated ducts survive and in the nipple the ducts and bundles of plain muscle-fibres are seen clearly. The cells are usually clearly packed and consist of lymphocytes and lymphoblasts, as well as dictocytic and syncytial reticulum cells (*Figs.* 52, 53). Mitotic figures are present, but small in numbers in view of the great cellularity of the tumour. Coarse bands of collagen form a supporting framework. The tumour cells reach the dermo-epidermal junction, and the overlying skin, although stretched, is intact. Fat and striped muscle on the deep surface of the breast are also infiltrated.

In view of this finding the previous biopsy was reviewed and the sections of the left radical mastectomy carried out in 1948 re-examined.

Final conclusion: the histological picture represented reticulosarcoma in both breasts.

DISCUSSION

Reticulosarcoma appears to be an apposite description of the histological picture of the sections examined in both breasts (*Figs.* 52, 53) since the tumour cells are not exclusively confined to cells of the lymphoid or reticulum series, but rather there is an admixture of mesenchymal cells with lymphocytes predominating. The multiplicity of names derived from the various descriptions of tumours of lymphoid tissue makes clinical comparison difficult. However, breast lesions with a similar histological picture appear to have been described by Droop (1940). Hill and Stout (1942) described 28 cases of sarcoma of the breast in 1900 malignant breast tumours. Four of these were lymphoblastic in nature.

The bilateral incidence of sarcoma of the breast appears to have been noted only twice previously in the literature. Harrington and Miller (1940) described lymphosarcoma of both breasts, and Adair, Craver, and Hermann (1945), in discussing Hodgkin's disease of the breast, cite a case by Kuckens in which the opposite breast was affected by the spread of the disease. In both these cases, however, the interval elapsing before the onset of the disease in the opposite breast was only a few weeks, whereas in the present case, one of the most unusual features has been the long lag of six years which elapsed before the right breast was affected. Perhaps, however, the most important clinical sign observed in this case has been the close resemblance of the lesion to carcinoma of the breast (*see Fig.* 51). Reddish discoloration of the skin, overlying a stony hard mass with *peau d'orange* and thickening of the nipple, all suggested epithelial neoplasia, more especially in the absence of any other clinical evidence of reticulosarcoma.

Prognosis.—A 5-year survival rate of 10 per cent for reticulosarcoma at all sites treated by surgery and radiotherapy is listed by Robb-Smith (1947) in a survey of reticulosarcomas and allied conditions. Harrington and Miller's patient with sarcoma of the breast survived only one year. Hill and Stout showed an average survival rate of 4–6 years, and a similar figure was quoted by Schreiner and Thibandeau (1932). The long interval elapsing before the other breast was affected in the present case and the absence of recurrence at the site of the first operation would suggest a more hopeful outlook.

The histological appearances afford no guidance as to whether the recent lesion was an autochthonous focus or metastasis, a differentiation which might influence the prognosis.

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A CASE OF PULMONARY METASTASES FROM CARCINOMA OF BREAST SUCCESSFULLY CONTROLLED BY TESTOSTERONE

By D. K. SAMBROOK, Swansea

Since the work of Loeser (1940) testosterone has become a well-established agent in the hormone treatment of advanced breast cancer, and has been found particularly valuable in controlling bone metastases. Soft-tissue metastases respond less often to this line of treatment, and even in the most favourable cases the regression of metastases seldom lasts more than two years.

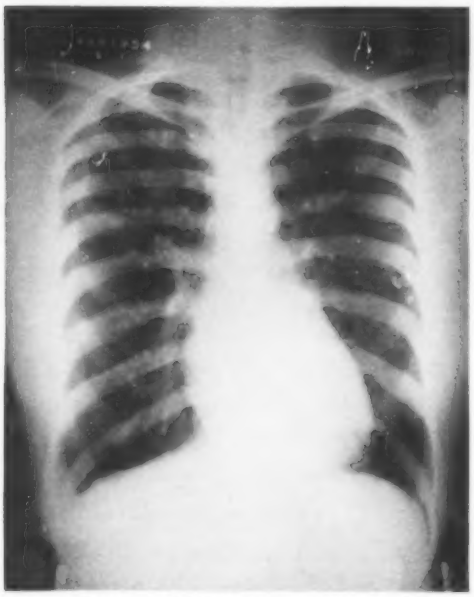

Fig. 54.—March, 1954. Multiple metastases in both lung fields, before testosterone therapy.

Fig. 55.—April, 1958. Lung fields remain clear of metastases. (Patient remains well, April, 1959).

The following case presents several interesting features: First, pulmonary metastases were caused to disappear by the administration of testosterone; secondly, no recurrence of these deposits occurred, although treatment was continued for less than one year; and finally, the tumour recurred at the primary site several years after treatment, but there was no recurrence of the pulmonary metastases.

CASE REPORT

The patient, a married woman aged 36 years, developed a lump in the upper inner quadrant of the left breast early in 1949. In September, 1949, she had local removal of the lump, which was found on section to be a scirrhous carcinoma of high-grade malignancy. It was decided to treat the breast and the regional glandular areas by deep X-ray therapy without any further surgical treatment. In October/November she received a tumour dose of 4000 r to the whole of the left breast, axilla, and supraclavicular triangle, in about 4 weeks. After the usual reaction the breast assumed a practically normal appearance, with only a small scar at the site of the tumour, with a little induration under it. The patient was followed up regularly and remained quite well until March, 1954, when she complained of a cough, and a radiograph of the chest showed the presence of several rounded opacities typical of pulmonary metastases (*Fig.* 54).

It was decided to induce menopause immediately by X-irradiation of the ovaries, and she was given methyl testosterone by mouth (in the form of linguets), 50 mg. twice a day, and this was continued for two months. At the end of that time she was feeling well, apart from some hot flushes; she had developed some acne on the face, presumably due to testosterone.

A radiograph of the chest taken in June, 1954, showed complete disappearance of the metastases seen in March. Further radiographs have been taken at intervals since then and none has shown any sign of reappearance of the metastases. A film taken in April, 1958, is shown (*Fig.* 55) for comparison with that taken in March, 1954.

In spite of this encouraging result, some doubt was felt regarding the efficacy of testosterone by mouth, so, in November, 1954, her doctor was requested to give her intramuscular injections of testosterone phenyl propionate, 100 mg. twice weekly for eight weeks. This was done. She has had no testosterone either by mouth or by injection since December, 1954, and she remained well, with no symptoms or signs of local recurrence or metastases for three years.

In February, 1957, the patient was referred to a gynæcologist because of dyspareunia. She was given

œstroform pessaries, which completely relieved this symptom. Each of these contained 0·1 mg. œstradiol benzoate. She used to insert one nightly for about ten days each month, and continued this until January, 1958. At this time she complained of some shooting pain in the scar in the left breast. A firm mass about 2 × 1 cm. was now palpable deep to the scar. This mass was at once excised. On section, it showed cords and clusters of active cancer cells embedded in a dense fibrous stroma.

It was decided not to proceed to any further radical treatment at this time. The previous irradiation precluded further treatment of this kind. We hesitated to perform radical mastectomy in view of the history of metastases in the chest. Careful follow-up examinations have been continued, and so far (April, 1959) there is no sign of further local recurrence, and radiography of the chest shows no sign of metastases.

COMMENT

Several interesting points are raised by this case. Complete disappearance of pulmonary metastases from breast cancer under the influence of treatment with œstrogens is not uncommon (Stoll and Ellis, 1953), but is less often seen with testosterone, and is usually only temporary in any case. In Stoll and Ellis's series only 2 out of 61 patients survived more than three years. It is remarkable that in this case metastases disappeared after only a few months' hormone treatment, and have not recurred more than three years later. It is also interesting that the tumour deposits in the lung were apparently unable to maintain their viability, whereas residual tumour cells in the breast must have done so. Presumably this is due to the different conditions in the tumour bed.

Another question that occurs to one is whether the administration of œstrogens, even in the small dosage and by the somewhat uncertain route of administration (per vaginam), in this case could be responsible for reactivating this evidently hormone-dependent tumour. The dose of œstrogens absorbed was insufficient to lead to uterine bleeding. To be on the safe side the patient was advised to discontinue the œstroform pessaries.

This case also prompts some reflections on the wisdom of relying too heavily on radiotherapy in the management of breast cancer. In recent years radical mastectomy has come under highly critical review, and there is legitimate dissatisfaction with the results of radical surgical treatment of breast cancer.

McWhirter (1950) has induced the Edinburgh surgeons to place their faith in radiotherapy to the extent of relying on it to deal even with such deposits —in axillary lymph-nodes—which could easily be removed technically. The preliminary results seem to support this policy. The remarkable capacity of breast cancer to lie latent, and yet recur years after quite drastic irradiation, is illustrated by this case, and may sound a note of caution.

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METASTATIC TUMOURS IN THE BREAST

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It is generally believed that secondary tumours in the breast are rare. While it is true that they are not common, experience in this department has shown that metastasis to the breast is more frequent than is usually supposed. The appearance of secondary tumour in the breast may be the first indication of tumour originating in the other organs; this is especially true of lymphomata and the leukæmias, but occurs also in carcinomata. Post-mortem examination of the breast in cases of malignant neoplasm will also sometimes reveal unsuspected metastatic tumour.

SURGICAL CASES

During an 11-year period (1947–57) 1304 specimens of primary malignant tumour of the breast, 412 specimens of benign tumour of the breast, and 7 cases of metastatic tumour in the breast were examined in this department. More recently, a further instance of almost certain metastatic tumour in the breast has been seen in this department. These eight cases and a ninth seen elsewhere deserve brief individual attention.

CASE REPORTS

Case 1.—This was a chloromatous mass removed from the breast of a 14-year-old girl who was later found to have an acute leukæmia. Leukæmic cells were aggregated in masses around the mammary ducts. This girl died shortly after biopsy of the breast.

Case 2.—A 28-year-old woman presented with a lump in the breast. The mastectomy specimen contained a well-circumscribed white mass 2·0 cm. in diameter which proved to be a well-differentiated malignant lymphoid neoplasm. Dense infiltrates were present around ducts and acini and extended into fibrous stroma and associated fat (*Fig.* 56). The axillary lymph-nodes were also found to be involved. After operation the breast wound healed slowly with obvious residual tumour at the base. Epistaxis, ecchymoses, and petechial formation were then noted. One month after mastectomy the white blood-count was 160,000 per c.mm.; many of the cells were monocytes. Later examinations showed more primitive and atypical cells of the monocyte series in the peripheral blood. Death occurred two months after mastectomy. Autopsy revealed generalized lymph-node enlargement, infiltration in lungs, stomach, spleen, bone-marrow, liver, kidneys, and contralateral breast. The infiltrating cells were of monocyte type and showed moderate mitotic activity.

Case 3.—A middle-aged woman, with a circumscribed lump in the breast, showed at mastectomy a 3·0-cm. diameter mass of appearance similar to that seen in *Case* 2. This proved to be a reticulosarcoma; again the axillary lymph-nodes were involved (*Fig.* 57). It was then discovered that the patient had had a mass of similar histological appearance removed from the orbit a short time before. The subsequent fate of this patient is not known.

Case 4.—This was a 45-year-old woman with a lump in the left breast. Some eighteen months previously a malignant cutaneous melanoma had been removed from the right heel. The breast mass measured 3·0 × 2·0 cm. and proved to be a secondary deposit of malignant melanoma (*Fig.* 58). At the time of writing, some 42 months later, this woman remains well and is carrying out her normal household duties. It may be mentioned here that in the period 1947–57 in this department two instances of primary malignant melanoma of the skin of the breast have been observed.

Case 5.—This woman, aged 40 years, complained of a lump in the breast for two weeks. This was sharply circumscribed, measured 1·5 cm. in diameter, and biopsy revealed a secondary anaplastic carcinoma of oat-cell type. Subsequent mastectomy showed no significant change in the remaining breast parenchyma or axillary lymph-nodes. Tomography demonstrated changes in the right main bronchus and smears made on bronchoscopic examination contained many cells of appearance consistent with origin in bronchial carcinoma. This patient's condition deteriorated rapidly; a palpable mass appeared in the abdominal wall and she died some six months after the appearance of the breast mass.

Case 6.—A 33-year-old woman presented with multiple subcutaneous masses in the muscles of trunk and limbs. A small discrete nodule was present in one breast. These nodules had been slowly increasing in size.

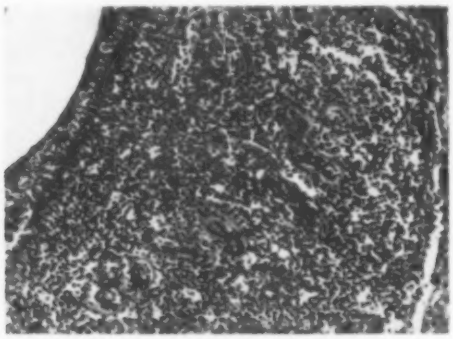

FIG. 56.—*Case* 2. Infiltrate of monocytes around a large duct in the breast of a 28-year-old woman. Hæmalum and eosin. (× 86·5.)

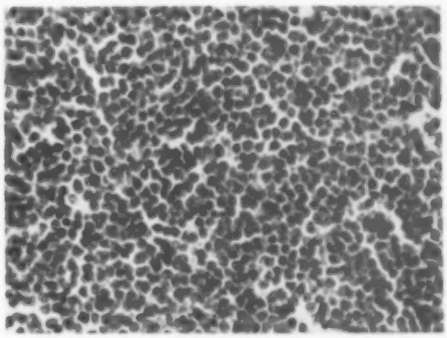

FIG. 57.—*Case* 3. Reticulosarcomatous mass in the breast of a middle-aged woman. Hæmalum and eosin. (× 240.)

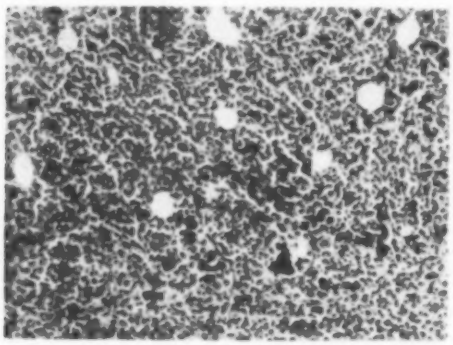

FIG. 58.—*Case* 4. Secondary malignant melanoma in the breast of a 45-year-old woman. Hæmalum and eosin. (× 86·5.)

FIG. 59.—*Case* 6. Leiomyosarcomatous nodule in the breast of a 33-year-old woman. Compressed breast parenchyma is seen to the left. Hæmalum and eosin. (× 86·5.)

Four years previously she had had a forequarter amputation for scapular leiomyosarcoma which infiltrated neck and axilla. Two years later a metastasis was removed from the rectus abdominis muscle. All the masses excised, including that in the breast, were leiomyosarcomatous. The breast nodule measured 1·25 cm. in diameter and showed some central necrosis. Pleomorphic nuclei and numerous mitoses were evident; the tumour nodule compressed the surrounding breast parenchyma (*Fig.* 59).

Case 7.—A 65-year-old woman complained of a lump in the upper outer quadrant of the left breast; this was fixed to the deeper structures only. About one year previously an inframammary plaque of carcinoma in situ of skin had been removed. The breast specimen contained a cystic mass of rather spheroidal cells 2·25 cm. in diameter. This mass was related to a group of small nerves which showed perineural lymphatic invasion. Some of the tumour cells were of squamous appearance. The tumour was considered to be secondary squamous carcinoma rather than a primary carcinoma of the breast. Shortly afterwards a tumour of the skin was removed from the patient's left lumbar region, but neither the nature

of this mass nor the subsequent fate of the patient is known.

Case 8.—This 56-year-old woman presented with a lump in the left breast. Radical mastectomy revealed a spherical, circumscribed mucoid tumour mass, 8·0 cm. in diameter, deep to the nipple. There was some skin ulceration medial to the latter. Histological examination showed this to be a mucoid carcinoma with well-formed acini of tall columnar epithelium present in the lakes of mucin (*Fig.* 60). The breast parenchyma was atrophic with no significant adenosis or epitheliosis. The axillary and internal mammary lymph-nodes showed no tumour,

FIG. 60.—*Case* 8. Mucoid adenocarcinoma in the breast of a 56-year-old woman who had a pyloric carcinoma. Hæmalum and eosin. (× 195.)

nor did the pectoral muscle. Biopsies of rib and iliac crest carried out at the same time were likewise free from secondary carcinoma. Oöphorectomy had also been performed; the cortical stroma was moderately cellular and no secondary carcinoma was seen.

The mastectomy wound failed to heal well and the patient's condition rapidly deteriorated. Severe vomiting developed, and two months after mastectomy a left pleural effusion became evident; gross œdema of the legs appeared followed by terminal pneumonia. Death occurred three months after mastectomy. At autopsy the patient was seen to be grossly emaciated and the mastectomy scar was incompletely healed. There was a right-sided basal pneumonia and pericarditis. A large annular malignant ulcer occupied the entire pyloric antrum. Histological examination showed this to be fairly well differentiated columnar-cell adenocarcinoma with areas of signet-ring cell extension and others of frankly mucoid appearance. Lymphatic permeation was evident; some arteries in the base of the ulcerated area showed obliterative change. The remaining breast showed slight adenosis and lipophagic change with calcification in some ducts. The endometrium was atrophic and cystic; the adrenals showed no medullary hyperplasia and there was vacuolation of many beta-cells of the anterior pituitary. This case is regarded as being almost certainly one of primary carcinoma of the stomach with secondary tumour in the breast, although the possibility of simultaneous development of mucoid carcinoma in both sites cannot be entirely excluded.

Case 9.—Dr. G. H. Smith of Maryfield Hospital, Dundee, has kindly informed me of a further instance of metastatic tumour in the breast. The patient, a 53-year-old woman, had a simple mastectomy for a lump in the right breast; the histological appearances were not typical of primary mammary carcinoma. Tumour later occurred in the sacral skin and bones. Despite radiotherapy, further skin nodules appeared; bilateral adrenalectomy was performed, but the patient died soon afterwards. Autopsy revealed a renal clear-cell carcinoma with multiple metastases. Review of the sections of breast showed an appearance consistent with metastasis from the renal carcinoma.

AUTOPSY CASES

The material studied comprised sections taken at consecutive autopsies from both mammary glands. In all, 70 cases of carcinoma of the breast and 148 cases of other malignant neoplasms in females, as well as 139 cases of malignant neoplasm in males, were studied. *Table I* shows the types of tumour studied and the number of cases showing breast metastases.

Study of the contralateral breast in 70 autopsies on cases of breast carcinoma revealed metastases in

Table I.—TYPES OF TUMOUR STUDIED AT AUTOPSY AND NUMBER OF CASES SHOWING BREAST METASTASES

PRIMARY TUMOUR TYPE	FEMALES		MALES	
	Total Cases	Number with Metastases in Breast	Total Cases	Number with Metastases in Breast
Mammary carcinoma	70	21 (30 per cent)*	1	0
Alimentary carcinoma	66	1 (1·5 per cent)	44	0
Respiratory carcinoma	23	2 (8·7 per cent)	45	1 (2·2 per cent)
Other carcinoma	25	0	15	0
Malignant melanoma	2	0	1	1 (100 per cent)
Sarcomata	12	0	8	0
Plasma-cell tumours	1	0	4	0
Lymphoma group	11	3 (27 per cent)	10	3 (30 per cent)
Leukæmias	6	3 (50 per cent)	11	5 (45 per cent)
Mycosis fungoides	2	0	0	0
Total	218	30 (13·8 per cent)	139	10 (7·2 per cent)

* Metastases to opposite breast.

30 per cent. In none of these was there fully convincing evidence that the tumour in the second breast was a further primary growth. In some cases the second breast did, however, show some duct epitheliosis not amounting to intraduct carcinoma. While it is not denied that bilateral primary breast carcinoma sometimes occurs, it seems very probable that in the majority of cases the tumour in the remaining breast is due to spread by the transthoracic lymphatics.

Other carcinomata metastasized infrequently to the breast. Probably carcinoma of the bronchus is the most likely to do so and involvement may be bilateral (*Fig.* 61). Metastases, however, were not uncommon in both male and female breasts in the lymphomata and leukæmias, especially the myeloid group. Metastatic infiltrates were noted in lymphatic, monocytic (*Fig.* 62), and eosinophilic leukæmia. Of the lymphomata, Hodgkin's disease most commonly affected the breasts; two of three females and both

males studied showed involvement of the breast. Mammary infiltrates were also seen in a 60-year-old woman with acute histocytic medullary reticulosis. Cases of lymphosarcoma, mycosis fungoides, and myelomatosis did not show breast infiltrates, with the exception of one male aged 36 years who showed periductal infiltrates of lymphosarcoma.

In general, metastases in the breast from carcinomata elsewhere are rather nodular, but where spread has occurred from the opposite breast the infiltration may be quite diffuse or sometimes produce irregular lumpiness. In the leukæmias infiltrates are often

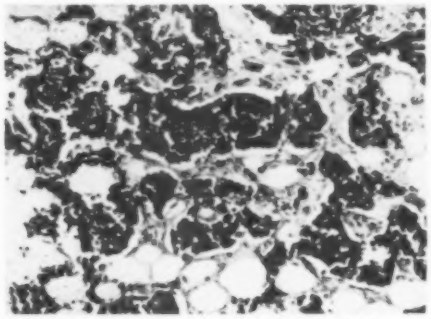

FIG. 61.—Anaplastic oat-cell carcinoma in the breast of a 50-year-old woman, discovered at autopsy. Hæmalum and eosin. (× 86·5.)

periductal or intralobular, but may affect both stroma and lobules. In Hodgkin's disease there are focal areas of infiltration mainly in the fibrous stroma. One woman, aged 30 years, died two years after the diagnosis had been established. During the last few weeks of life the breasts had slowly enlarged and at autopsy were grossly swollen, pendulous, and globular in shape, with some purplish discoloration of the overlying skin. On section they were markedly œdematous and the axillary lymph-nodes were moderately enlarged. Histological examination revealed focal nodules of pleomorphic-celled tissue; bizarre giant cells, eosinophils, and some fibrosis were noted. Similar infiltrates were seen in lymph-nodes, bone-marrow, pericardium, pleura, liver, and ovaries, but not in the spleen.

Here it must be emphasized that non-specific periductal infiltrates are not uncommon in the female breast. In younger women intralobular infiltrates also occur. In my autopsy series of 800 consecutive cases (Sandison, 1957), 186 showed such infiltrates, i.e., 23·2 per cent of cases. Of these, 21·0 per cent showed lymphocytes only, while 2·2 per cent also had plasma cells present. The highest incidence falls in the 26–30 years age-group; plasma cells occur most often also in the younger age-groups. Periductal lymphocyte infiltrates are much less common in the male; in my series of 500 consecutive males (1957), periductal lymphocyte infiltrates were seen in only 24 cases, an incidence of 4·8 per cent. In general, only older males show such infiltrates.

DISCUSSION

It is generally accepted that metastatic tumours in the breast are not common. In a recent publication Dawson (1958) states that the breast is one of the rarer recorded sites of metastatic tumour growth, although it may sometimes be involved in the terminal stages of generalized malignant conditions, including melanomatosis and leukæmia. She points out that where mammary carcinoma is bilateral the tumour in the second breast may be metastatic. Dawson has personally studied two metastatic tumours in the breast, one from a gastric carcinoma and the other from a uterine leiomyosarcoma. The former case she has described in detail (1936); both breasts contained metastatic tumour.

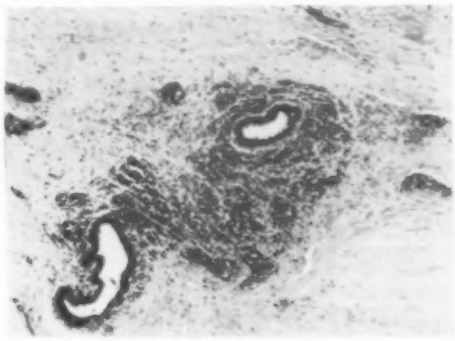

FIG. 62.—Periductal infiltrates of monocytes in the breast of a 77-year-old male with monocytic leukæmia discovered at autopsy. Hæmalum and eosin. (× 56·5.)

Tumour metastasis to the breast has been considered by Willis (1952). Involvement of the breast by lymphomata has been discussed by Schultz-Brauns (1933), Geschickter (1945), Seifert (1952), and Haagensen (1956). Haram (1937) has described in detail a case of lymphatic leukæmia with bilateral mammary involvement treated by mastectomy. Anderson and Roberts (1954) have described a chloromatous mass in the breast of a 16-year-old girl with acute monocytic leukæmia.

Charache (1942, 1953) has discussed tumour metastasis to the breast in greater detail and has described 10 cases personally studied at the Brooklyn Cancer Institute since 1942. These include 3 cases of cutaneous melanoma, 2 ovarian and 2 renal carcinomata, and 1 case each of thyroid, prostatic, and uterine corporeal carcinoma. Including these cases and others culled from the literature he has assembled 25 cases of tumour metastases in the breast. The primary sites are stomach 5, ovaries 4, cutaneous melanoma 4, kidney 3, cervix uteri 2, and 1 each from liver, prostate, corpus uteri, thyroid, mediastinum, os pubis, and opposite breast.

Charache claims that metastatic tumours in the breast are less fixed to the surrounding tissues than primary growths and may often be freely mobile. Pain is never present. In his experience the tumour size varies from 1·0 to 4·0 cm. in diameter.

It is obvious from the present findings that metastatic tumours in the breast are commoner than is sometimes supposed.

Probably many cases of metastatic tumour in the breast are not reported. Charache certainly underestimates the frequency with which mammary carcinoma metastasizes to the opposite breast; this is, in the female, by far the commonest source of

secondary tumour in the breast and is probably almost invariably the result of transthoracic lymphatic spread. Bilateral mammary carcinoma in the male is rare; no case has been seen in this department, but Treves and Holleb (1955) report an incidence of 2·7 per cent in their series of 146 cases.

In other instances where the primary tumour is not in the breast it seems likely that blood-spread is an important factor. Nevertheless, the possibility of distant metastasis in any organ of the body by lymphatic spread is probably underrated.

Routine examination of the breasts at tumour autopsies would greatly increase the known incidence of metastatic tumour in the breast. We have noted in this department that the incidence of metastatic carcinoma of the breast in pituitary, ovaries, adrenals, thyroid, and the opposite breast is higher than previously supposed, when careful search for these is made at autopsy and in surgical specimens removed for, and in the control of, ablative endocrine treatment for breast carcinoma.

It must, however, be conceded that in the only other large series of autopsy breast examination (225 cases) reported by Frantz, Pickren, Melcher, and Auchincloss (1951) no report is made of the finding of metastatic tumour.

The other feature of interest which emerges in the present findings is the relative frequency of deposits in the breasts in cases of diseases of the leukæmia and lymphoma group in both males and females. This is not surprising in view of the nature of these neoplastic processes. Indeed, involvement of the breast may sometimes be the presenting symptom in such cases.

SUMMARY

Metastatic tumours in the breast are not so rare as is commonly supposed. Surgically removed specimens of such tumours have been examined in one case of chloroma, monocytic leukæmia, reticulosarcoma, malignant melanoma, carcinoma of the bronchus, leiomyosarcoma of peripheral origin, squamous carcinoma, and probably gastric carcinoma. A further case from a renal carcinoma is also reported.

In the female breast, at autopsy, the commonest metastatic tumour is from the opposite breast (30 per cent of cases of breast cancer coming to autopsy). In 148 other female tumour post-mortem examinations, metastases in the breast have also been noted from carcinoma of the bronchus (2), carcinoma of the pancreas (1), acute leukæmia (3), Hodgkin's disease (2), and acute histiocytic medullary reticulosis (1).

In 139 tumour post-mortem examinations in males the following metastatic tumours in the breast have been noted, viz., malignant melanoma (1), carcinoma of the bronchus (1), chronic myeloid leukæmia (2), monocytic leukæmia (2), eosinophilic leukæmia (1), and Hodgkin's disease (2).

Acknowledgements.—My thanks are due to Dr. G. H. Smith for the notes on his case of metastatic renal carcinoma in the breast, and to Professor C. F. W. Illingworth, Mr. E. Gerstenberg, Mr. A. B Kerr, Mr. W. Sillar, and Mr. G. Russell Thomson for permission to quote their cases. Mr. G. Kerr and Mr. W. Mason took the photographs.

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PROLAPSE OF THE LIP MUCOSA

BY MICHAEL C. OLDFIELD, LEEDS

OCCASIONALLY, the mucosa of the upper lip dips below the free margin of the vermilion border, in the form of an ugly and annoying fold (*Fig.* 63). This arises usually, I think, as a congenital malformation, but may be acquired by habitual indrawing of the lining of the lip through a gap between the natural teeth or an ill-fitting denture. In time a small prolapse gradually becomes a larger one, because the suction causes œdema and hypertrophy. The swollen lining of the lip is drawn inwards between the teeth, then when the suction force is released, the loosened mucosa prolapses below the free margin of the lip.

CASE REPORTS

(7 *Cases*)

1. Congenital Type (5 cases).—The congenital type usually affects the upper lip, and is bilateral and symmetrical.

Case 1.—G. C., male, aged 25 years. Prolapse of mucosa on each side of lower lip since birth. Operation, Dec. 30, 1946.

Case 2.—B. P. W., male student, aged 17 years. Prolapse of mucosa on each side of upper lip since birth (*Fig.* 64). Operation, Jan. 9, 1950.

Case 3.—S. H., miner, aged 24 years. Prolapse of mucosa on each side of upper lip since birth. Operation, Aug. 11, 1950.

Case 4.—R. A. Y., schoolboy, aged 12 years. Prolapse of mucosa on each side of upper lip since birth (*Fig.* 65). Operation, Sept. 7, 1954.

Case 6.—B. W., soldier, aged 17 years. When 8 years old his left upper lateral incisor tooth was knocked out when he fell from a bus. Afterwards he developed the habit of sucking his lip through this gap, and gradually the lining of his upper lip on the left side has increased in

FIG. 63.—*Case* 5. Before and after operation. Congenital prolapse of the mucosa of the upper lip which has been present since birth, in a married woman aged 24 years.

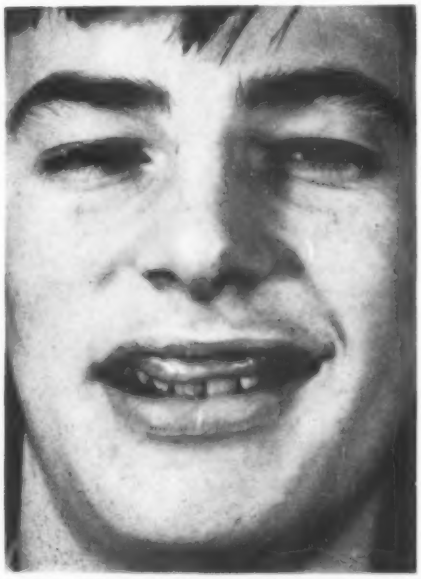

FIG. 64.—*Case* 2. Congenital prolapse of mucosa of the upper lip in a male student aged 17 years.

FIG. 65.—*Case* 4. Congenital prolapse of mucosa of the upper lip, since birth, in a schoolboy aged 12 years.

Case 5.—R. M., housewife, aged 24 years. Prolapse of mucosa on each side of upper lip since birth (*Fig.* 63). Operation, Aug. 25, 1958.

2. Acquired Type (2 cases).—The acquired type seems to affect the upper or lower lip, and is generally unilateral.

size and projects below the vermilion border and causes disfigurement (*Fig.* 66). Operation, Oct. 27, 1950, excision of elliptical area of redundant mucosa on the left side of the upper lip.

Case 7.—G. L., widow, aged 83 years. For the last two years since the removal of a mucous cyst from the left

side of the lower lip she has acquired the habit of sucking the lining of the lower lip between the cutting edges of her dentures, which do not come into proper occlusion on the left side. No operation advised (1958).

TREATMENT

The results of treatment of the prolapse of the upper lip seem to be very good, but the lower lip

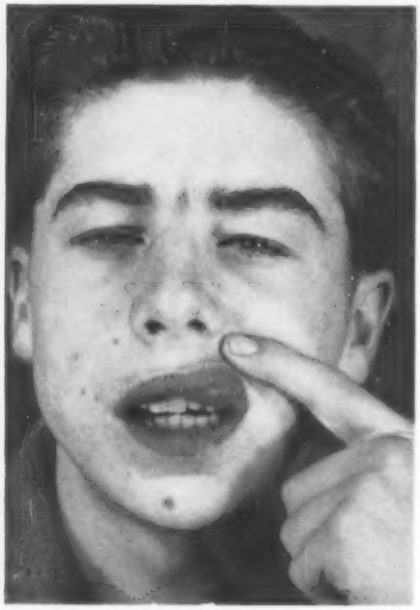

FIG. 66.—*Case* 6. Acquired prolapse of the mucosa of the left side of the upper lip in a soldier aged 17 years.

is more difficult to treat, especially in old folk, with dentures that are unstable due to atrophy of the gums.

OPERATION

(*Fig.* 67)

Under an intratracheal anæsthetic, the lip is everted by the assistant's fingers and the redundant mucosa is marked with Bonney's blue. An elliptical excision of mucosa and submucosa is made. After the bleeding has been stopped, the defect is repaired by 'end-on' mattress sutures of chromic catgut; the first bite passes through the orbicularis oris muscle as well as the mucosa, whereas the second bite only includes the mucosa. Care must be taken to avoid an ugly bulge at each end. If this should occur, the redundant mucosa at each end is excised.

No special after-treatment is required, but the patient is warned that the effect of the operation

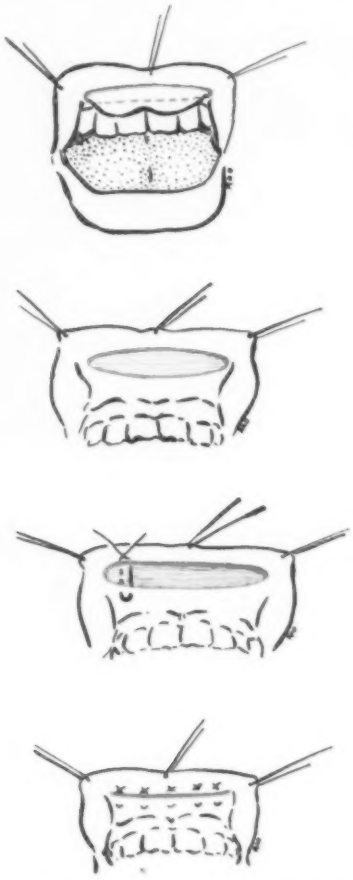

FIG. 67.—Operation for repair of congenital prolapse of the mucosa of the upper lip.

cannot be judged until all œdema has subsided, and this may take 3–4 weeks. The patient goes home on the day following operation, and should be seen again for review after 6 weeks.

RADIO-ACTIVE IMPLANTATION OF THE PITUITARY

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In the Western Infirmary during the past four years, three methods of implanting radio-active material into the sella turcica have been used to destroy the pituitary gland in patients with advanced malignant disease—radon-seed implantation (Forrest and Peebles Brown, 1955; Forrest, Peebles Brown, Morris, and Illingworth, 1956); yttrium-90 (Y^{90}) rod implantation (Forrest, Blair, Morris, Peebles Brown, Sandison, Valentine, and Illingworth, 1958); and Y^{90} screw implantation (Forrest, Blair, and Valentine, 1958). Further assessment of their associated complications, effectiveness in destroying the pituitary, and clinical value is reported here.

METHODS

Clinical Assessment.—Cortisone maintenance therapy (50 mg. per day) was usually started a few days after radio-active implantation of the pituitary. Thyroid replacement (thyroxine, 0·3 mg. per day) was given when the patient developed clinical signs of myxœdema, usually in two to three months. Each patient was seen by at least two of us at monthly intervals, and their state assessed on clinical and radiological grounds. Patients were regarded as benefiting from treatment only when there was uncontroversial evidence of regression of all manifestations of cancer. Specific inquiry was made at each visit for visual upset, rhinorrhœa, and diabetes insipidus. Thirty-six patients treated by radon implantation and 36 by Y^{90} rod implantation had detailed examination of the visual fields before and after treatment (Harrington, 1958a, b). Thereafter, examination of the fields was carried out only when the patient complained of visual upset.

Extent of Destruction of Pituitary.—The destructive effect of the radio-active implant on the pituitary gland has been estimated by histological examination. At autopsy, after removing the brain and dividing the pituitary stalk, the diaphragma sellæ was incised peripherally, and the posterior wall of the sella removed. The complete hypophysis was eased out of the sella by gentle blunt dissection without damaging the capsule, and at once fixed in 10 per cent formol saline. Care was especially necessary in removing glands which had been transfixed by Y^{90}-loaded screws. When firm, the gland was divided horizontally at three to five sites, depending on its depth. The slices were placed in corrosive-sublimate-formol overnight, processed, and each sectioned at several levels. Sections were stained routinely with hæmalum-eosin, phosphotungstic acid hæmotoxylin, and the periodic acid-Schiff technique alone, and by Pearse's method. An approximate estimate of the extent of destruction was made on examination of representative sections. Destruction was regarded as being synonymous with total loss of cell nuclei and outlines. Chromatin granules may take up stain for some time after cell death, especially those of the alpha cells. Recognizable cells with nuclei were regarded as being viable since it is not certainly known that cells of altered appearance do not continue to produce hormone.

RESULTS

Radon Implantation.—Forty-five patients with advanced malignancy (36 of breast origin) were treated by the implantation of 10–20 mc. radon into the pituitary fossa. In the first 25 patients a single cannula was passed through the right nostril and two to four 5-mc. seeds introduced; in the others two cannulas were inserted, one through each nostril, and a single 8-mc. seed introduced into each half of the fossa.

Complications.—

Mortality: No patient died within 24 hours of operation.

Visual upset: Progressive loss of vision followed implantation of radon seeds in 8 of the 45 patients. In 4 patients this did not develop until 3–12 months after implantation (*Table I*). With one exception, visual loss, once it occurred, progressed relentlessly to total loss of sight. The delay in onset and the histological changes in the optic chiasma at autopsy indicated that visual disturbance was due to the effect of necrosing radiations and not to trauma during implantation. Two patients developed partial III nerve palsies.

Table I.—Visual Damage in 8 Patients after Pituitary Radon Implantation*

Case No.	Dose Radon (mc.)	Time of Onset after Implant
R4	15	Under 1 wk.
R6	20	
RMY	15	
R9	15	3 wk.
R22	16	3 mth.
R8	20	5 mth.
R17	15	7 mth.
R3	15	15 mth.

* In 7 patients visual loss was finally complete.

Diabetes insipidus: Excessive thirst and polyuria were common after radon implantation, although spontaneous improvement tended to occur in 3–6 months. Pitressin therapy was frequently required.

Rhinorrhœa: Leakage of cerebrospinal fluid from the nose developed in 2 patients, 2 and 6 months after implantation (16 mc.). In one patient widespread cerebral metastases were present and progression of these caused death. The other patient had recurrent attacks of meningitis which proved fatal.

Extent of Destruction of Gland.—The extent of necrosis of the pituitary following implantation of various doses of radon was estimated post mortem

in 20 patients (*Fig.* 68). Complete necrosis of the gland was found in only one patient, in whom radiation necrosis was present also in the hypothalamus. In the others, necrosis varied from 10 to 90 per cent of the gland depending on the dose of radon and position of the seeds.

The necrotic area was always sharply demarcated from apparently viable tissue. The appearance of the affected cells varied according to the time after implantation. They stained deep red, with nuclei which were at first pyknotic, and later disintegrated, only chromatin dust remaining. Chromophil granules were detected in the necrotic area for a long time after implantation, but eventually cytoplasmic remnants disappeared and only a collagenous stromal network remained. A reactive polymorph infiltrate was frequent, but active organization of necrotic tissue was never seen. The changes in the vascular spaces included vasculosis, thrombosis, and hæmorrhage.

Clinical Results.—Regression of cancer of the breast followed radon implantation in 10 of 36 patients treated, while one additional patient had arrest of disease (*Tables II, III*).

None of the 9 patients with other malignancy (4 malignant melanoma, 2 squamous carcinoma of the cervix and vulva, 1 carcinoma of the body of the uterus, 1 sarcoma of the breast, 1 multiple myelomatosis) had any benefit from pituitary radiation with radon.

Yttrium-90 Rod Implantation.—Fifty-three patients with metastatic breast cancer and one patient with metastatic chorion-epithelioma of the testis were treated by implanting two rods of irradiated yttrium oxide (50 mg.; 6·3 × 2·3 mm.) into the pituitary fossa, the total dose varying from 8 to 17

A

CASE No.	TOTAL DOSE mc	RADIOLOGICAL POSITION Lat.	RADIOLOGICAL POSITION A.P.	IMPLANT TO DEATH Days	TRANSVERSE SECTION OF HYPOPHYSIS	ESTIMATED DESTRUCTION
Rm1	10·0			241		80 %
R10	10·0			26		30 %
R 1	10·0			28		30 %
Rm3	10·0			44		30 %
R 2	10·0			516		20 %
R 12	10·0			84		15 %

B

CASE No.	TOTAL DOSE mc	RADIOLOGICAL POSITION Lat.	RADIOLOGICAL POSITION A.P.	IMPLANT TO DEATH Days	TRANSVERSE SECTION OF HYPOPHYSIS	ESTIMATED DESTRUCTION
Rm2	15			22		90 %
R 4	15			1072		60 %
R 17	15			370		50 %
R 16	15			46		30 %

C

CASE No.	TOTAL DOSE mc	RADIOLOGICAL POSITION Lat.	RADIOLOGICAL POSITION A.P.	IMPLANT TO DEATH Days	TRANSVERSE SECTION OF HYPOPHYSIS	ESTIMATED DESTRUCTION
R 8	20			256		100 %
R 7	20			254		60 %
R 15	15			104		15 %

D

CASE No.	TOTAL DOSE mc	RADIOLOGICAL POSITION Lat.	RADIOLOGICAL POSITION A.P.	IMPLANT TO DEATH Days	TRANSVERSE SECTION OF HYPOPHYSIS	ESTIMATED DESTRUCTION
R 23	16			71		80 %
R 24	16			31		80 %
R 26	16			17		70 %
R 36	16			87		70 %
R 31	16			251		66 %
Rm9	16			22		20 %
R 32	16			11		10 %

FIG. 68.—Extent of destruction of the pituitary in 20 patients after implantation of radon seeds. A, Single cannula, 2 seeds; B, Single cannula, 3 seeds; C, Single cannula, 4 seeds; D, Two cannulas, 2 seeds. White circles show position of seeds in gland. Black area—destroyed, stippled area—surviving tissue.

mc. In each patient the sources were inserted by means of twin cannulas, one through each nostril. The rods were prepared by a method similar to that described by Rasmussen, Harper, and Kennedy (1953).

Finely powdered spectrographically pure yttrium oxide (Y_2O_3) was mixed with DPX mountant to form a smooth thick paste which was compressed into a thick-walled capillary tube, 30 × 2·5 mm. internal diameter, until a suitable length (about 10 mm.) was obtained. This was extruded onto a glass slide by means of a steel rod which fitted exactly into the capillary tube. The rods were dried at 100–110° C. for 2 hours (after which they could be handled with reasonable care) and transferred to a slip-cast alumina crucible. The subsequent firing was carried out in stages. After heating to 800–900° C. in a gas-fired muffle for 1–2 hours, the crucible was placed inside another of graphite, covered by an alumina lid and heated gradually in a Phillips high-frequency generator to 1650–1700° C. and this temperature held for 10–15 minutes. The sintering which occurred during this treatment caused a slight contraction to 2·25–2·3 mm. diameter, and the resulting rods were strong enough to withstand normal handling.

The rods were cut to weight 50 mg. (6·3 mm.), coated with nobecutane (Evans), and dispatched for activation in the Harwell pile BEPO.

Table II.—Radon Implantation in Cancer of the Breast*

Effect on Cancer	No. of Patients	Average Duration of Life following Implantation (Weeks)
Progressed	25	12
Stationary	1	163
Regressed	10	87

* Results of treating 36 patients with advanced breast cancer by radon implantation of the pituitary between January, 1955, and February, 1956. All are now dead.

Complications.—

Mortality: Two patients whose general condition was very poor died within 24 hours of implantation.

Visual damage: Only 5 patients complained of disturbance of vision, of whom 4 had detailed examination of the field loss (*Fig.* 69). The defects were of minor significance, and, unlike those following radon, did not progress. One patient who developed such a defect (Ys 1) has remained well for nearly 3 years without increase in its extent.

Diabetes insipidus: Temporary polyuria after implantation was common, but this was rarely severe and never persisted for more than 3 months. Only 2 patients required pitressin therapy.

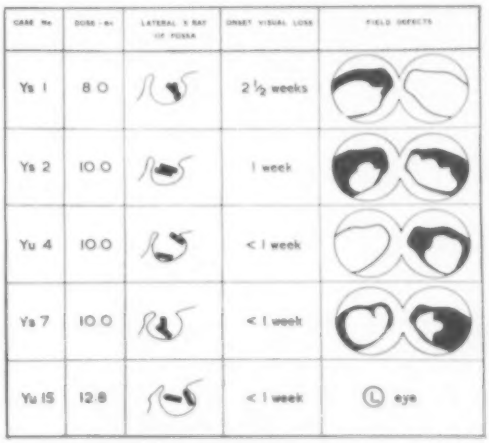

Fig. 69.—Extent of visual damage in 5 patients following Y^{90} rod implantation of the pituitary.

Rhinorrhœa: A leak of cerebrospinal fluid through the nose developed in 10 patients 1 to 4 weeks after implantation, and led to recurrent attacks of meningitis. These usually responded to systemic and intrathecal penicillin with systemic sulphadiazine. Three patients were treated by packing muscle into the pituitary fossa at craniotomy (Mr. Sloan Robertson). One patient lived for 17 months following this procedure without recurrence of the leak; the other 2 died. Two patients were treated by inserting a

Table III.—Regression of Cancer following Implantation of the Pituitary with Radon

Case No.	Age (Years)	Lesions undergoing Regression	Duration of Life (Weeks)
		Regression of disease following Pituitary Radon Implant—10 *Cases*	
R2	48	Skeletal metastases	74 (adrenalectomy 39 wk.)
R3	49	Primary tumour, lymphatic and skeletal metastases	131 (adrenalectomy 52 wk.)
R4	35	Lymphatic and skeletal metastases	153
R6	53	Pulmonary metastases	119
R7	62	Local recurrence, skin nodules, skeletal metastases	107
R8	63	Local recurrence, skin nodules, skeletal metastases	38 (hypothalamic coma)
R9	54	Skeletal metastases	107
R17	66	Primary tumour, skin nodules, lymphatic metastases	53
R22	40	Lymphatic and skeletal metastases	47
R31	45	Skeletal metastases	36 (meningitis)
		Arrest of disease following Pituitary Radon Implant—1 *Case*	
R20	50	Primary tumour, local infiltration, lymphatic metastases	163 (re-implant 157 wk.)

Y^{90}-loaded screw into the holes previously made in the fossa which stopped the leak in both. One patient has remained well for 10 months after this second implantation, the other died in 2 weeks from

FIG. 70.—Patients with rhinorrhœa following Y^{90} rod implantation of the pituitary.

progression of cancer. The other 5 patients with rhinorrhœa died at home—in 2 meningitis appeared to be responsible.

In 3 patients rhinorrhœa resulted from over-penetration of the fossa during insertion of the cannulas. In 6 of the remaining 7 patients one or both rods were positioned in the anterosuperior angle of the fossa, suggesting that necrosis of the diaphragm sella in this area was primarily responsible for the leak (*Fig.* 70).

Extent of Destruction.—The necrosing effect of two rods of Y^{90} on the normal pituitary has been assessed in 23 patients post mortem (*Fig.* 71). Destruction was complete in 4 patients. Of the remainder, 11 had 90–99 per cent and 8 under 90 per cent of the gland destroyed. In 2 of the latter (Ax 6, Yu 5), death took place within 24 hours of implantation.

	TOTAL DOSE mc	RADIOLOGICAL POSITION Lat.	RADIOLOGICAL POSITION A.P.	IMPLANT TO DEATH	TRANSVERSE SECTION OF HYPOPHYSIS	ESTIMATED DESTRUCTION
Ys 7	10.0			118 days		99 %
Ys 3	10.0			555 days		99 %
Yu 1	16.0			4 days		95 %
Yu 8	10.0			20 days		95 %
Ys 15	11.7			83 days		95 %
Ym 2	13.9			169 days		95 %
Ys 20	13.9			302 days		95 %
Yu 16	11.3			5 days		90 %
Ys 11	10.0			10 days		90 %
Yu 10	11.7			267 days		90 %
Ys 14	10.0			304 days		90 %

B

CASE No.	TOTAL DOSE mc	RADIOLOGICAL POSITION Lat.	RADIOLOGICAL POSITION A.P.	IMPLANT TO DEATH	TRANSVERSE SECTION OF HYPOPHYSIS	ESTIMATED DESTRUCTION
Yu 7	13.0			8 days		100 %
Yu 12	12.9			13 days		100 %
Ax 5	11.3			34 days		100 %
Yu 18	11.2			85 days		100 %

A

CASE No.	TOTAL DOSE mc	RADIOLOGICAL POSITION Lat.	RADIOLOGICAL POSITION A.P.	IMPLANT TO DEATH	TRANSVERSE SECTION OF HYPOPHYSIS	ESTIMATED DESTRUCTION
Yu 11	14.1			50 days		80 %
Ax 21	11.2			44 days		75 %
Ax 9	15.0			8 days		65 %
Ys 4	10.0			168 days		60 %
Ys 10	8.5			126 days		50 %
Yu 13	10.9			143 days		50 %
Ax 6	12.9			1 day		30 %
Yu 5	9.5			0		0 %

C

FIG. 71.—Extent of destruction of the pituitary in 23 patients after implantation of Y^{90} rods. A, Destruction complete, 4 cases. B, 90–99 per cent destruction, 11 cases. C, Under 90 per cent destruction, 8 cases. White circle shows position of rods in gland. Black area—destroyed, stippled area—surviving tissue.

The histological changes in the pituitary following Y^{90} implantation were essentially similar to those seen following radon implant, although occasionally in patients surviving for a long time necrotic material was replaced by pale staining hyaline connective tissue, sometimes surrounding empty cystic spaces.

Clinical Results.—The value of Y^{90} rod implantation of the pituitary as a palliative treatment for advanced breast cancer was assessed in a series of 57 patients, in whom either pituitary implantation or adrenalectomy and oöphorectomy was performed. The choice of treatment was selected by a random

Table IV.—PITUITARY Y^{90} IMPLANTATION COMPARED WITH ADRENALECTOMY AND OÖPHORECTOMY*

METHOD OF TREATMENT	NO. OF PATIENTS	REGRESSION OF CANCER			NO. OF PATIENTS STILL IN REMISSION
		No. of Patients	Duration of Regression	Duration of Life	
Pituitary Y^{90} rod implantation	28	9	75 wk.	80 wk.	4
Adrenalectomy and oöphorectomy	32	14	73 wk.	80 wk.	3

* Patients treated between October, 1955, and December, 1956, and assessed December 31, 1958.

Table V.—REGRESSION OF CANCER FOLLOWING (*A*) IMPLANTATION OF THE PITUITARY WITH Y^{90} RODS, AND (*B*) ADRENALECTOMY AND OÖPHORECTOMY

CASE NO.	AGE (YEARS)	LESION UNDERGOING REGRESSION	DURATION (WEEKS)	
			Regression	Life
		A. Regression following Pituitary Y^{90} Implantation—9 Patients		
Ys1	53	Local recurrence, soft tissue metastases, pleural effusion	141 +	141 +
Ys3	58	Ulcerating local recurrence, lymphatic metastases	47	79 (adrenalectomy 79)
Ys6	49	Primary tumour, lymphatic and skeletal metastases	125 +	125 +
Ys8	52	Other breast and lymphatic metastases, ascites	114 +	114 +
Ys13	57	Skin nodules, lymphatic, peritoneal, and skeletal metastases	64	72
Ys14	57	Skin nodules, other breast, lymph-nodes, pulmonary and skeletal metastases	33	43
Ys16	47	Primary tumour, lymphatic, pulmonary, and skeletal metastases	19	19
Ys20	66	Primary tumour, skin nodules, lymphatic and pulmonary metastases	46	46
Ys22	60	Tumour both breasts, lymphatic metastases	82 +	82 +
		B. Regression following Adrenalectomy and Oöphorectomy—14 Patients		
1	71	Ulcerating local recurrence, lymphatic metastases	13	13
4	44	Pleural effusion, skeletal metastases	148 +	148 +
6	65	Ulcerating local recurrence, lymphatic metastases	46	54 (pituitary implant 54)
7	61	Primary tumour, lymphatic metastases	141 +	141 +
9	51	Primary tumour, lymphatic and skeletal metastases	76	80 (pituitary implant 79)
11	65	Lymphatic and skeletal metastases	91	91
15	62	Pleural effusion, lymphatic and skeletal metastases	100	102
17	60	Ulcerating local recurrence, skeletal metastases	70	96 + (pituitary implant 80)
23	51	Mammary and lymphatic metastases	67	75 +
24	49	Skin nodules, pleural effusion, lymphatic metastases	74 +	74 +
25	58	Local recurrence, skin nodules	64	70 +
26	30	Skin nodules, mammary, lymphatic, peritoneal, and skeletal metastases	50	65 +
27	55	Local recurrence, skeletal metastases	50	50
30	46	Skin nodules, mammary, lymphatic, and pulmonary metastases	31	54 + (pituitary implant 40)

method in patients judged suitable for either operation. Three patients initially selected for treatment by adrenalectomy were found at operation to have metastatic deposits around the adrenals which prevented their removal. These three patients were subsequently treated by pituitary implantation, and thus included in both groups. Nine of 28 patients treated by implantation of the pituitary had definite and worth-while regression of disease compared with 14 of 32 patients treated by adrenalectomy and oöphorectomy. The duration of regression and of life in those patients who responded in the two groups was similar (*Tables IV, V*).

Fifteen patients who, by virtue of advanced disease or poor general condition, were unfit for adrenalectomy and oöphorectomy, and therefore not included in the above trial, were treated by implantation with Y^{90}. Only 3 patients had short-lived benefit (20, 38, and 68 weeks).

Eight patients previously treated by adrenalectomy and oöphorectomy, 6 without benefit, were subsequently also treated by pituitary implantation with Y^{90}, but without response. The remaining patient treated by Y^{90} rod implantation was an acromegalic with breast cancer. She remains well 2½ years after implantation. There has been improvement in visual field defects which were present before implantation.

A male patient with chorion-epithelioma of the testis and pulmonary metastases who had not benefited from orchidectomy and adrenalectomy had no response after implantation of the pituitary with Y^{90} rods.

Table VI.—Total Cases Treated by Y^{90} Rod Implantation of the Pituitary

Patients Treated	No.	Regression of Cancer
Breast cancer		
Included in series	28	9
Unfit for adrenalectomy, not treated in series	16	3
Previous adrenalectomy	8	0
Acromegaly	1	1
Chorion-epithelioma testis	1	0

These results are summarized in *Table VI*.

Yttrium-90 Screw Implantation.—This modified technique was introduced to achieve accurate placing and fixing of the two sources of Y^{90} in an optimum position in the sella. Each source is encased in nylon and fitted into a small stainless steel base which is screw-threaded. The Y^{90} sources are inserted through a drill hole so that the threaded base is gripped in the anterior wall of the sella while the active rod projects into the pituitary gland (*Fig.* 72). One screw is inserted into each half of the gland under radiological control with an image intensifier. A detailed description of the method of insertion of the screws has already been published (Forrest, Blair, and Valentine, 1958).

Experience of this technique in 42 patients (38 with advanced malignancy) has confirmed that it permits precise and consistent placing of a radioactive source in the centre of each half of the gland. This method of treatment was reserved for patients who on account of poor general condition were considered unsuitable for adrenalectomy and oöphorectomy, or who had previously been treated by adrenalectomy and oöphorectomy or Y^{90} rod implantation. In 5 patients who were too ill for general anæsthesia, implantation was carried out following topical application of 10 per cent cocaine. (*Table VII.*)

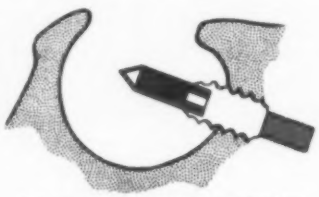

Fig. 72.—Diagram of Y^{90} screw implant.

Table VII.—Patients treated by Y^{90} Screw Implantation of the Pituitary. February, 1958, to January, 1959

Disease	Dose Y^{90} (mc.)	No. of Patients
Carcinoma breast	13·8–14·5	31
" "	9·0–9·2	6
Carcinoma pancreas	13·8	1
Diabetes mellitus	14·0	1
Acromegaly	14·6, 18·4, 29·2	3

In the first 33 patients with normal pituitaries the total activity for each pair of rods (5 × 2 mm.; 65–70 mg.) was 13·8–14·5 mc. In 6 patients recently treated this dose has been reduced to 9·0–9·2 mc., each source measuring 3 × 2 mm. (45 mg.). In 3 acromegalics the total dose of Y^{90} was 14·6–29·2 mc.

Complications.—

Mortality: Three very ill patients died within 24 hours of screw implantation. In one the operation was performed with local anæsthesia. Advanced disease in 5 other patients was responsible for death within 2 weeks of implantation.

Visual upset: Two patients noticed deterioration in the visual acuity of one eye 2 weeks and 5 months after implantation. The sources of Y^{90} in both patients were well placed in the centre of the fossa, the total dose being 14·0 mc. The visual defects have not progressed (*Fig.* 73). A third patient developed an extra-ocular nerve palsy in the right eye 3 days after implantation; one source was placed laterally in the fossa (*Fig.* 74).

Diabetes insipidus: Severe thirst and polyuria have been the rule after Y^{90} screw implantation with the larger dose (13·8–14·5 mc.) and pitressin therapy has been required in all patients. Diabetes insipidus has not been severe when the smaller dose of Y^{90} (9·0–9·2 mc.) has been used, and pitressin has not been required.

Rhinorrhœa: Rhinorrhœa and/or meningitis occurred in 3 patients following the implantation of 13·8–14·5 mc. Y^{90} by the screw technique (*Fig.* 75). In one case (S 10), a patient with cerebral metastases, the floor of the sella collapsed during implantation and the posterior clinoid was

penetrated. At autopsy a large defect was found in the floor of the sella.

In the other 2 patients the sources were placed centrally without complication. Both died from examined post mortem (*Fig.* 76). At the most, surviving tissue was limited to small groups of subcapsular cells, which can have been of little functional significance.

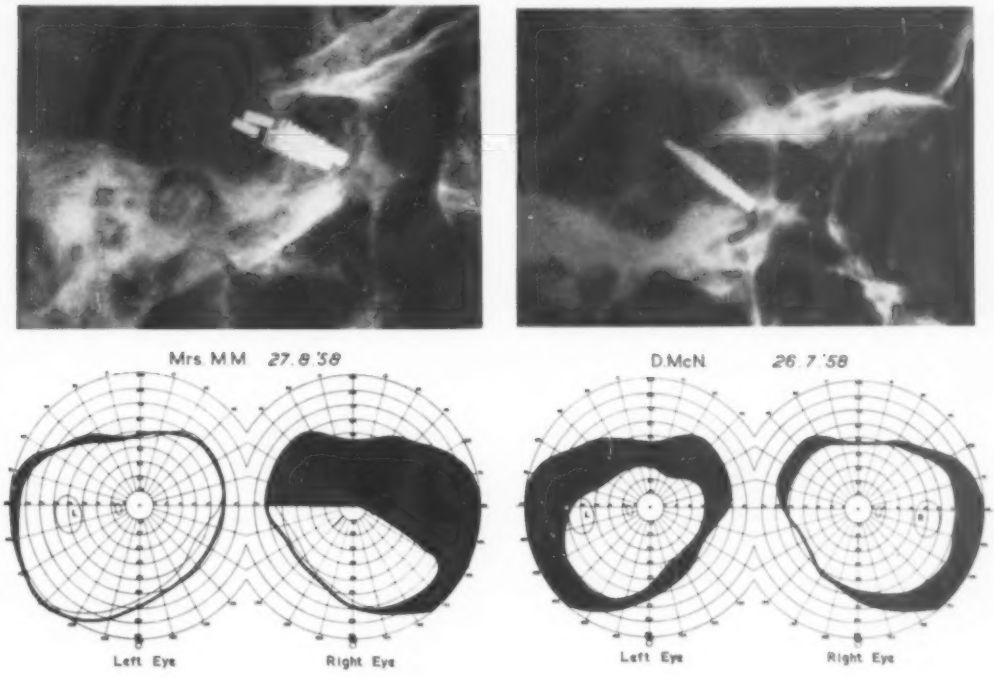

FIG. 73.—Visual defects in 2 patients following Y^{90} screw implantation of the pituitary. A, *Case* S7, 14·0 mc. onset, 5 months. B, *Case* S18, 14·0 mc. onset, 2 weeks.

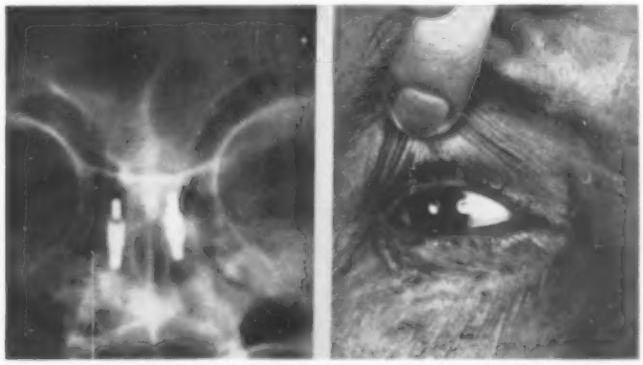

FIG. 74.—Third-nerve palsy in a patient following Y^{90} screw implantation of the pituitary (*Case* S4, 13 mc.). Note lateral position of one screw.

pneumonococcal meningitis, the organism being similar to those cultured from the contents of the sphenoidal sinus. In these 2 patients at autopsy one screw was found to be loosely gripped by the bone.

Extent of Destruction.—Destruction of the pituitary has been virtually complete in all 12 patients

Clinical Results.—The advanced disease and poor general condition of the patients in this group have prevented worth-while assessment of the therapeutic value of this modified technique. Only 3 patients have had lasting objective regression of cancer and remain well 20, 44, and 45 weeks after implantation. Ten patients are not yet assessed.

DISCUSSION

The implantation of radio-active material into the pituitary fossa is technically simple, but if the hypophysis is to be destroyed completely and without complication great care must be taken in placing sources of suitable type.

Radon seeds proved entirely unsuitable, as their penetrating gamma rays (maximum energy 1·8 MeV.) irreparably damaged the optic chiasma in 8 of 12 patients who survived for over 6 months following their implantation. Despite the delivery of a substantial dose of destructive radiation to the surrounding tissues, complete destruction of the pituitary was found in only 1 of the 20 patients in whom the extent of necrosis of the gland was assessed histologically post mortem, and in this patient advanced radiation changes were also present in the hypothalamus. In the 19 patients with incomplete destruction, a zone of necrosis extended 3–4 mm. from each source and was clearly demarcated from surrounding gland of normal histological appearance. The similarity of this pattern to that following implantation of radon in a *glass* tube (Lacassagne and Nyka, 1934), grains or rods of gold 198 (Au^{198}) (Young, 1957), and rods of Y^{90} suggests that the necrotic zone is the result of electron rather than gamma radiation. A source of gamma rays thus needlessly endangers surrounding structures.

Similar complications after radon implantation have been described by other workers (Westminster Hospital Report, 1956; Weinbren, Jackson, and Gamsu, 1957), and radon seeds should no longer be used for this purpose.

Screened grains and unscreened rods of radio-active gold (Au^{198}) have also been implanted into the pituitary by transnasal routes (Talairach and Tournoux, 1955, 1958; Talairach, Aboulber, Tournoux, and David, 1956; Greening, 1956, 1957, 1958; Ellis, Jackson, Lewis, Nias, and Oliver, 1958), and transethmoidal routes (Bauer, 1956 a, b; Bauer and Schweitzer, 1958). This isotope also emits both beta particles and gamma rays (maximum energy of β 0·96, γ 0·41 MeV.), its equivalent activity in terms of gamma radiation dose being approximately one-fifth that of radon (1 mc. radon = 4·8 mc. Au^{198}; Sinclair, 1952). Complete destruction of the pituitary is rarely achieved by gold implantation (Young, 1957; Bauer and Schweitzer, 1958) and the large doses required to produce significant destruction (55–60 mc.; Denoix and Juret, 1958; Ellis and others, 1958) endanger the optic chiasma.

CASE No.	DOSE – mc	ONSET WEEKS AFTER IMPLANTATION	MENINGITIS	PROGRESS	LATERAL X RAY OF FOSSA
S 10	14·5	4	+	Overpenetration During Insertion. Died – 6 weeks	
S 6	14·0	5	+	Died – 6 weeks	
S 33	13·8	3	+ No Rhinorrhœa	Died – 3 weeks	

FIG. 75.—Rhinorrhœa and or meningitis following Y^{90} screw implantation of the pituitary.

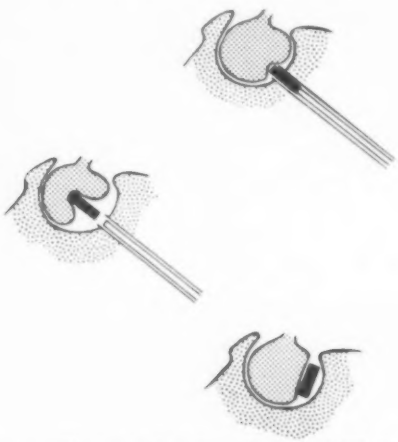

FIG. 77.—Diagram to show the main cause of difficulty in placing free sources of Y^{90} in the pituitary gland.

CASE No.	TOTAL DOSE mc	RADIOLOGICAL POSITION		IMPLANT TO DEATH Days	TRANSVERSE SECTION OF HYPOPHYSIS	ESTIMATED DESTRUCTION
		LATERAL	A.P.			
S 2	13·8			1		99%
S 12	14·5			4		99·9%
S 21	14·0			4		98%
S 8	14·5			7		99%
S 1	13·8			8		99%
S 34	12·6			19		98%
S 33	13·6			22		100%
S 6	14·0			43		100%
S 28	14·0			81		100%
S 10	14·5			84		100%
S 4	13·6			90		98%
S 3	13·6			357		100%

FIG. 76.—Extent of destruction of the pituitary in 12 patients after Y^{90} screw implantation. White circle shows position of seeds in gland. Black area—destroyed, stippled area—surviving tissue.

Radio-active materials emitting only beta particles of high energy are more likely to effect radiation necrosis limited to the pituitary gland. A satisfactory source must also have a suitable half-life and be available in a solid form. These requirements are met by Y^{90} which emits only beta particles of 2·2 MeV., has a half-life of 64 hours, and can be prepared in a form suitable for implantation by activating sintered rods of yttrium oxide.

As the maximum range of its beta particles in soft tissue is about 1 cm., well-placed sources of correct dose should not irradiate the optic chiasma, and in our patients visual defects followed Y^{90} rod implantation only when a source was situated in the upper part of the fossa. Unlike that following radon implantation the visual disturbance was of little consequence and did not progress.

The extent of pituitary destruction resulting from the implantation of Y^{90} in our series of 23 post-mortem examinations is similar to that found by other workers using free sources of Y^{90}, whether these are introduced transnasally (Fergusson, 1957; Dogliotti and Ruffo, 1958; Moseley, Ironside, and Harper, 1958; Greening, 1959; Notter, 1959) or transcranially (Kelly, Evans, Harper, and Humphreys, 1958). Correlation of the radiological position of the rods with the extent of destruction effected in our cases indicates that the implantation of two 6 × 2-mm. rods of total dose 11 mc., one in the centre of each half of the gland, can completely destroy the pituitary without complication. None of the 4 patients with complete destruction had either visual disturbance or severe diabetes insipidus.

In our experience free rods of Y^{90} cannot be consistently placed with such ideal precision (*Fig.* 77). On the other hand, the modified technique of screw implantation allows controlled placing of the sources in an optimum position and is regularly followed by virtually complete destruction of the gland.

A serious complication of transnasal intrasellar implantation of free rods of Y^{90} was persistent rhinorrhœa which was frequently complicated by meningitis. This complication has also been described by other workers with Y^{90} (Fergusson, 1957; Greening, 1957; Dogliotti and Ruffo, 1958; Moseley and others, 1958; Fraser, Joplin, Laws, Morrison, and Steiner, 1959); Au^{198} (Ellis and others, 1958; Talairach and Tournoux, 1958), and radon (Westminster Hospital Report, 1956). Its incidence following implantation of radon and Au^{198} is less than that when Y^{90} is used. Recent histological studies have clearly demonstrated that the diaphragma sellæ completely closes the roof of the fossa except for an opening for the pituitary stalk (Mahmoud, 1958). The subarachnoid space does not normally extend into the sella turcica, and the space between the pituitary gland and the bone is normally filled only by fibrous tissue and vascular spaces. Rhinorrhœa can therefore occur only if the diaphragma is breached, and its association with a Y^{90} source lying in the anterosuperior area of the fossa indicates that radiation necrosis of the nearby diaphragma is primarily responsible for the leak. At this point there is no intervening gland tissue to protect the membrane from the necrosing radiations of the Y^{90}.

A similar relationship between rhinorrhœa and high-placed sources has been recently described by Fraser and others (1959), and these workers have found that its incidence is reduced by placing the sources more than 2·5 mm. from the diaphragma. The delay in onset in the patients who developed rhinorrhœa following radon implantation also suggests that it is due to a radiation effect.

Rhinorrhœa following transnasal insertion of radio-active sources into the sella can be prevented by plugging the holes in the anterior wall through which they are introduced. Talairach and Tournoux (1958), have successfully used small tantalum screws for this purpose, and other workers have used agar powder (Ellis and others, 1958), solutions of sodium alginate and calcium chloride (Fraser and others, 1959), and 'spongestan' cylinders (Notter, 1959). In our opinion the most important single factor in preventing the occurrence of rhinorrhœa is accurate placing of the sources at a safe distance from the diaphragma sellæ. Such accurate placing is readily accomplished by screw-implantation, which has the additional safeguard of plugging the holes in the anterior sella wall.

The occurrence of rhinorrhœa in 2 patients following screw implantation (13·8–14·5 mc.), despite uncomplicated and accurate placing of the sources, indicates that with this dose sufficient beta radiation is delivered to the diaphragma to cause damage. The high incidence of diabetes insipidus and the development of visual defects in 2 of the 33 patients in whom a similar dose was accurately placed denotes supradiaphragmatic radiation, and also indicates that the dose of Y^{90} is too large. The occurrence of 99 per cent destruction of the gland in one patient within 24 hours of inserting 13·8 mc. of Y^{90} by the screw technique is clear evidence that this dose is larger than required, as approximately one-quarter of the activity only would be expended in this time. Since reducing the total dose to 9·2 mc. severe diabetes insipidus, visual disturbance, or rhinorrhœa have not occurred. The smaller size of the sources has the added advantage of allowing a greater proportion of the screw to project into the fossa, giving a firmer grip on the bone.

It is difficult to calculate accurately the dose of beta-emitting material required to destroy the pituitary without delivering significant radiation to surrounding structures, but it has been estimated that at 5 mm. from a spherical source of 5 mc. the dose would be in the region of 3,000,000 rads. The magnitude of this dose confirms the fact that the pituitary gland is highly radio-resistant. It is our present endeavour to find the minimum dose of Y^{90} required to produce complete destruction. This is more likely to be achieved by practical observations of the extent of necrosis effected by known doses of Y^{90} than by theoretical calculations.

The clinical value of pituitary implantation in the treatment of advanced breast cancer compared with other forms of endocrine ablation cannot yet be assessed, as the methods hitherto used have been followed by severe and sometimes fatal complications and do not regularly produce complete destruction of the pituitary gland. The trial we carried out in a comparable series of patients suggested that the implantation of free rods of Y^{90} into the pituitary

fossa did not offer as good a chance of palliation as adrenalectomy and oöphorectomy. The evidence we now present indicates that the implantation of fixed sources of Y^{90} by the screw technique can regularly achieve uncomplicated destruction of the gland comparable in extent to that following combined surgical and radiation techniques (Au^{198} in fibrin; Radley Smith, 1957; Baron, Gurling, and Radley Smith, 1958: Y^{90} in wax; Edelstyn, Gleadhill, Lyons, Rodgers, Taylor, and Welbourn, 1958). To date this method has been used only in those patients who were considered unsuitable for, or previously treated by, adrenalectomy and oöphorectomy. A trial of this procedure in more favourable cases now appears justified.

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OBSERVATIONS ON EXTRADURAL HÆMORRHAGE

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INTRODUCTION

THE following observations on extradural hæmorrhage were prompted by reading the 1927 edition of Rose and Carless's *Textbook of Surgery* which was in current use during my undergraduate days. This volume gives a very clear and concise account of the 'classical' development of an extradural hæmorrhage, but it is stated that the "prognosis is extremely unfavourable".

Although the pathology of the extradural hæmorrhage had been described and the need for surgical intervention recognized much earlier, it was not until late in the nineteenth century that a more hopeful view of the prognosis was taken. Jonathan Hutchinson (1867), in his concluding remarks on "Effusion of Blood between Bone and Dura Mater", stated: "It is a remarkable fact that the modern annals of surgery do not, as far as I am aware, contain any cases in which life has been saved by trephining for this state of thing." Eighteen years later a much more hopeful view was taken by Jacobson (1885–6) who, in an admirable review of 71 cases of extradural hæmorrhage, found that 10 patients survived, a mortality-rate of 86 per cent. Forty-two years later '*Rose and Carless*' (1927) quoted Von Bergmann as stating that of 99 patients only 16 recovered, the mortality-rate still being 86 per cent.

Table I.—EXTRADURAL HÆMORRHAGE WITH MORTALITY-RATE EXCEEDING 30 PER CENT

Author	*No. of Cases*	*Mortality per cent*
Bradley (1951)	27	56
Goinard and Descuns (1948)	43	42
Larghero (1955)	13	76
Lazorthes (1952)	12	58
Munro and Maltby (1941)	44	55
Pringle (1938)	33	66
Rowbotham, Maciver, Dickson, and Bousfield	27	37
Voris (1947)	22	45
Woodhall, Devine, and Hart (1941)	177	56

With the development of neurosurgery as a special field for surgical endeavour, one would expect a fall in the mortality-rate, but for some years head injuries were not frequently admitted to neurosurgical clinics, nor were neurosurgeons much concerned with their management, and the mortality-rate still remained high. During and after the second world war neurosurgeons took an increasing interest in head injuries, and it was felt that there had been a considerable change in the prognosis of extradural hæmorrhage. When recent publications were reviewed it was apparent that many still accepted a mortality-rate for this condition which was far too high. In a comparable surgical text-book Aird (1957) states that the mortality-rate is about 50 per cent, and it is true that many series reported in the past twenty years show similar figures (*Table I*).

On the other hand, there have been a few reports with much lower mortality-rates, as shown in *Table II*.

In view of the disparity between these figures it was felt that a review of a larger series of patients who developed extradural hæmorrhage would be worth while. Thus it was the assessment of the mortality-rate which initiated this survey of 83 cases of extradural hæmorrhage encountered in the past eleven years in the neurosurgical service of a large adult general hospital and a children's hospital.

Table II.—EXTRADURAL HÆMORRHAGE WITH MORTALITY-RATE LESS THAN 30 PER CENT

Author	*No. of Cases*	*Mortality per cent*
Gurdjian and Webster (1942)	34	27
Ingraham, Campbell, and Cohen (1949)	20	10
Jamieson (1954)	12	8
Lewin (1954)	46	21
Makela (1950)	12	25
Rowbotham (1949)	33	21
Sartorius and Humphries (1946)	20	5
Whalley (1958)	30	17

The series is a personal one inasmuch as all the patients have been operated upon by the author or by house-surgeons under direct supervision. Other cases occurred in the hospitals included in this survey, and the results are comparable with those appearing in this paper; they are not included because some of the factors to be discussed require detailed observation which was not always recorded. In this survey of the mortality-rate, other features of the condition which seemed worthy of record were inevitably introduced. A few observations upon extradural hæmorrhage thus became a more complete review of the present position as the condition is seen and treated today.

Extradural hæmorrhage is an acute surgical emergency which must be taught so clearly and concisely to students that, should they meet the condition ever so infrequently in later years, the possibility of its presence must arise in their minds and the implications and necessary treatment must be realized.

AGE INCIDENCE

Extradural hæmorrhage is rare as a result of birth injury. Only one such complication occurs in this series and it was associated with a shallow depressed fracture in the parietal region; it gave rise to no specific manifestations although it measured 6 cm. across and was 1 cm. deep. A search of the literature reveals only a few isolated cases with this complication of birth injury (Ballance and Ballance, 1922; Lefkowitz, 1936), and few cases in newborn babes are found in published series of extradural hæmorrhages occurring in childhood. Ingraham, Campbell, and Cohen (1949) found no such cases in their series of 20 children. Gurdjian and Webster (1942) found 2 cases resulting from birth injury. A recent report by Douglas (1957) reveals one such hæmorrhage in a series of 8 children.

There is still much confusion among statements concerning the frequency of this condition in later

childhood. Ingraham, Campbell, and Cohen showed that the complication occurred in 1·8 per cent of head injuries in children requiring admission to hospital. Certainly this figure is less than the 3 per cent usually quoted for adults, but it is hardly sufficiently low to justify the comment made by Johnson (1954) concerning the "obvious infrequency of the typical extradural hæmorrhage in children".

However, the statement that it is rare in the first two years of life has some support. In this series

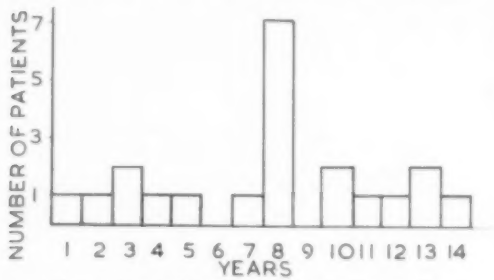


FIG. 78.—Age incidence in 21 children up to 15 years of age, showing a peak incidence at 8 years and the paucity of cases in the first two years of life.

only 2 of the 21 children under 15 years of age were less than 2 years of age, and one of these cases resulted from a birth injury, as did the only case under 2 years of age in the series reported by Douglas. The condition was most frequent between the sixth and tenth years—7 in this series were 8 years of age (*Fig.* 78). These figures are at marked variance with those reported by Ingraham, Campbell, and Cohen, who found 11 of their 19 cases in infants under 2 years of age. They attribute this finding to the loose attachment of the dura to the bones of the skull in the early years, together with the greater vascularity of the meningeal vessels. These conditions cease to operate after the age of 8 years. The discrepancy between the two series in this respect is difficult to reconcile without involving differences in the selection of material.

In adult life, the peak incidence is in the third decade. Almost one-quarter of all the patients were in this age-group (*Fig.* 79). The oldest patients in this series were 67 years of age. Thus it would seem that no age is immune and that there is little significant difference in any decade up to the fifth, when the frequency declines sharply.

The nature of the injury in relation to the age-group was of little interest, except to show that, in childhood, falls on the head were more frequent causes than traffic accidents.

Ingestion of alcohol was a significant factor in the production of this complication in the middle-age group in the same manner as that observed in head injuries generally.

NATURE OF THE INJURY

Extradural hæmorrhage is rarely found as a complication of compound fractures of the vault of the skull, although it is not uncommonly associated with those internal compound fractures involving the base of the skull. In this series 3 cases were associated with fractures into the frontal sinus and 3 with the middle ear. In general, extradural hæmorrhage is seen as a complication of blunt head injuries of the closed type. It is of interest to ascertain the proportion resulting from traffic accidents as distinct from other peace-time causes, i.e., blows upon the head, falls on the head. When these figures are compared with the series of 71 cases reported seventy-four years ago by Jacobson (1885–6) it is found that traffic accidents head the list, while in 1886 they were almost insignificant. In Jacobson's series the causes of this complication may be grouped as follows:—

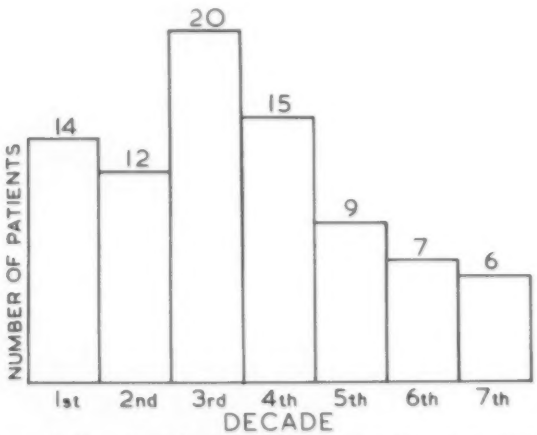


FIG. 79.—Age incidence for all decades, showing the peak incidence in the third decade.

1. Blows upon the head, 18 per cent. These included 3 patients who were struck by a musket ball which did not penetrate the skull.

2. Falls upon the head, 70 per cent. This group includes 3 patients who fell from slowly moving

vehicles and which could not be called traffic accidents in the modern sense.

3. Traffic accidents, 7 per cent. Of these, 2 patients were knocked down by horse-drawn vehicles and 2 by trains.

In the present series 39 per cent of cases were involved in traffic accidents, 36 per cent were the result of falls, while 16 per cent followed blows upon the head, a figure comparable to that observed in Jacobson's series. The cause of the head injury was not ascertained in 8 instances. The relative proportions observed in the two series are illustrated in *Fig.* 80.

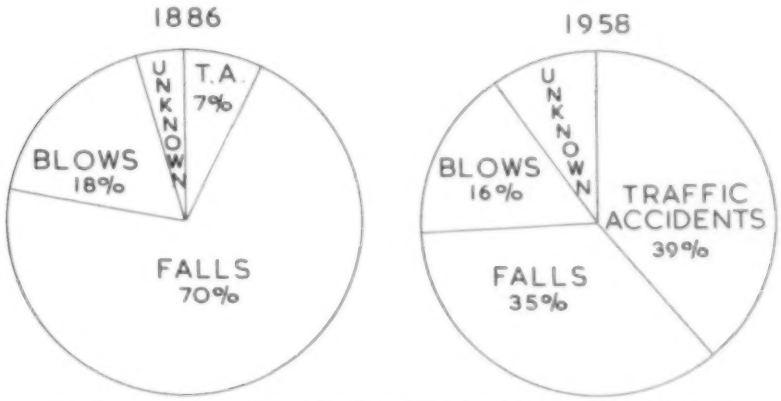


FIG. 80.—A comparison between the nature of the injury in the series reported by Jacobson in 1886 and the author's series.

SEVERITY OF THE INJURY

It has been generally accepted that the blow which ultimately results in extradural hæmorrhage is not usually a severe one, but this statement originated in the days before high-speed travel became commonplace and resulted in a great increase in the number of head injuries due to road accidents. It is, therefore, of interest to ascertain whether there should be any modification of this statement in the light of these changes. Information about the exact nature of the impact on the head is most frequently lacking in motor accidents, but an assessment can be made from the duration of the initial unconsciousness. Adequate information concerning this period was available in 27 patients. The injury was not severe enough to cause initial unconsciousness in 6, while in 8 the initial period of unconsciousness was less than 30 minutes, the average time being about 10 minutes. In 7 the initial period of unconsciousness was estimated to be greater than 1 hour but less than 6 hours. The remaining 6 patients remained unconscious until operated upon, and of these, 3 were considered to have an impact of only moderate severity and the failure to regain consciousness was due to the presence of the extradural clot and not to the brain injury. Three patients sustained severe injuries but only one of these died. Thus, of the 27 patients in whom adequate information was available, 14 sustained impacts which were of mild degree. In 10 these were of moderate severity (*Table III*).

There is less difficulty in assessing the severity of the impact following a fall. In Jacobson's series, which still remains the best documented series recorded, of the 44 patients in which a fall caused the head injury, 16 fell from a standing position and the distance through which the head fell was less than 6 ft., and the velocity of impact could be said to be less than 13 miles per hour (19 ft. per second). Sixteen patients fell distances between 6 and 12 ft., and in these the velocity of impact would be less than 17 miles per hour (25 ft. per second). In 11 only, the height of the fall exceeded 12 ft. In the present series most falls were less than 8 ft.

Table III.—SEVERITY OF INJURY IN TRAFFIC ACCIDENTS—27 CASES

	Unconscious Period	*No. of Cases*	*Percentage*
Mild	None	6	52
	Less than 30 min.	8	
Moderate	1–6 hr.	7	37
	Until operation	3	
Severe	Injury—operation	3	11

Of those who sustained blows upon the head, only 4 were rendered unconscious and only 1 of these sustained a severe injury.

This analysis confirms the often repeated statement that extradural hæmorrhages follow relatively mild injuries. It is not clear why more severe injuries do not sustain large extradural hæmorrhages. McKenzie (1938) states that there is no satisfactory explanation for this clinical finding, but suggests that the blood-pressure and the œdema of the brain which occur in a severe injury promote rapid coagulation in a ruptured dural vessel. It is probable that the injury produces a potential extradural space in which the extravasated blood accumulates. This extravasation may be limited by depression of bone on the one hand—extradural hæmorrhage is uncommon in the presence of a depressed fracture—or by cerebral swelling and intradural hæmorrhage

on the other. If the intradural hæmorrhage should continue in the presence of either of these conditions, the patient is likely to die at an early stage before the extravasation reaches a size comparable with those encountered in the less severe generalized brain injury. This explanation appears satisfactory in many, but certainly not in all, cases.

SITE OF IMPACT

It is to be expected that the extradural hæmorrhages will be more frequent when fractures cross the lines of the meningeal vessels. Gurdjian, Webster, and Lissner (1953) have shown that fracture lines which are directed across the middle meningeal vessels, lateral or sigmoid sinuses, follow impacts of low velocity on the lateral aspect of the head. The correlation between the site of impact and the fracture line is so good that the direction of the fracture can be predicted if the site of impact is known. This principle is of great importance in the provisional diagnosis of extradural hæmorrhage.

Thus, the site of impact is of the greatest importance in the determination of the occurrence of the extradural hæmorrhage. This is so true that every recipient of a blow in the temporoparietal region must be suspected as a potential candidate for an extradural hæmorrhage. Although in one patient it appeared that the extradural hæmorrhage had been produced by the prolapse of a cortical vessel through a dural tear, the remaining cases all arose from branches of the middle meningeal vessels, from the dural sinuses, or from diploic channels in the skull. In one patient the relation between the site of injury and the meningeal vessel was unique. This patient, who had had a subluxation of the cervical spine, became deeply comatose with bilateral fixed dilated pupils some hours after the application of skeletal traction to the skull. At operation it was found that the drill had torn a large middle meningeal artery and this had resulted in the development of a large extradural clot.

Signs of external injury to the head are usually present, although on occasions the scalp had to be shaved to reveal abrasions clearly. In a very large proportion of cases the injury to the scalp was related to the site of the hæmatoma. In 70 of the patients the abrasion or hæmatoma of the scalp directly overlay the extradural clot. In 2 patients only could the signs of external injury have been misleading, while in 11 patients only was it not possible to obtain any indication of the site of the clot because no sign of external injury was present at the time the operation was performed. Thus it was found that extradural hæmorrhage in the anterior fossa was accompanied by signs of impact in the frontal region, while laterally placed injuries in the temporal and parietal regions were followed by a hæmorrhage in the middle fossa or parietal region. Posterior fossa clots followed blows on the posterior third of the head. In some respects this external indication of injury was a more reliable guide than the radiological evidence of a fracture line, for a fracture in the region of the pterion could be associated with a clot in the middle fossa or in the anterior fossa, but in almost all instances the site of the impact overlay the clot, although the fracture line may have been placed more towards the periphery.

ORIGIN OF THE HÆMORRHAGE

The extravasation may originate from the middle meningeal arteries or veins, from dural venous sinuses, or occasionally from diploic veins, but there appears to be a considerable difference of opinion regarding the most frequent source. Amongst British writers there is a tendency to accept statements that the hæmorrhage usually arises from venous sources, and Wood-Jones (1912 a) is usually quoted as the source of this information, but he was not dogmatic upon this point. He states: "One would be inclined to suspect that the greater majority are caused by bleeding from easily torn sinuses." He then remarks that it is in the slowly developing hæmatomata that the bleeding is likely to be venous. In a later paper Wood-Jones (1912 b) again emphasizes the venous origin of the hæmorrhage on the basis of three post-mortem examinations in which injection and later serial section failed to reveal a rupture of the artery. The first two cases presented were of relatively slow onset and the information on this point is inadequate in the third. So it might reasonably be expected that these would be venous in origin. Paul (1955) stated that in 33 consecutive cases the bleeding was always of venous origin. On the other hand, Voris (1947), in 22 patients, found the middle meningeal artery to be the source in 14. Two arose from the lateral sinus, while 6 were probably venous in origin. Lazorthes (1952), in 12 patients, found 8 to be of arterial origin, while LeCount and Apfelbach (1920), in a series of 199 extradural hæmorrhages found at autopsy, considered that 93 were of arterial origin.

In this series the site of origin of the hæmorrhage was not always made clear in the earlier cases, but in the last 34 consecutive cases the origin was observed to be arterial in 15, in 5 it arose from both artery and vein, and tears in the dural venous sinuses occurred in 8. In the remaining 6 the origin was not specifically stated in the operation note, or no satisfactory single point of origin could be found. It is probable that some of these were due to tearing of the middle meningeal veins alone, and it must be admitted that in many of the cases in which the middle meningeal artery or one of its branches was torn there was usually a general ooze from small or minute tributaries of the veins, which were either stripped off from the bone at the moment of impact or later by the accumulation of blood-clot.

Table IV.—Site of Origin of Bleeding in relation to Time between Injury and Operation

Origin of Bleeding	*No. of Cases*	*Average Time between Injury and Operation*
Venous	8	3½ d.
Mixed arterial and venous	5	24 hr.
Arterial	15	8½ hr.

The interval between injury and operation is of interest when compared with the source of bleeding, as shown in *Table IV*.

It would seem that there is a good correlation between the rate of development of the syndrome and the rate, or rather the origin, of the bleeding.

DEVELOPMENT OF THE HÆMATOMA

The production of a large hæmatoma in the extradural plane from a small bleeding-point which

is sometimes venous in origin raises some problems. Does the accumulating mass of blood-clot strip the dura from the bone, or does the bleeding occur into a potential extradural space which has been produced by mechanical distortion of the skull and dura at the moment of impact?

The strength with which the dura adheres to the inner surface of the skull varies from place to place

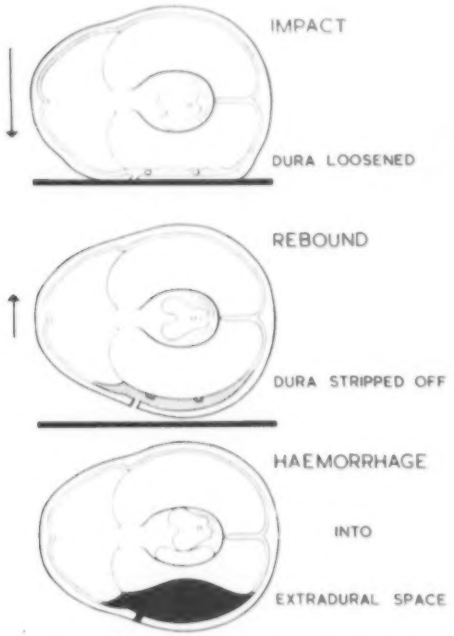


FIG. 81.—The formation of an extradural space by the stripping of the dura from the bone following impact and rebound.

and from patient to patient, a fact which makes generalizations difficult. Bell (1816) suggested that the inbending of the skull at the time of injury, followed by the outward deflexion of the skull, strips the dura away from the inner surface of the calvarium, and it is into this space that the hæmorrhage takes place (*Fig.* 81).

That this deformation of the skull takes place is obvious when studies of the mechanical effects of head injuries are undertaken. When the moving skull meets a hard surface its kinetic energy is transformed into the deformation of the skull. This energy may be dissipated to a greater or lesser extent by fracturing of the skull. If there is no fracture, the skull tends to rebound as the energy stored up in the deformation is again released as kinetic energy. This rebound phenomenon is an important factor in the production of head injuries and is often considerable. *Fig.* 82 was obtained from a strip of 16-mm. cine-film taken during an amateur screen play during which one of the actors slipped from the bonnet of a car, striking his head on the roadway. Five hours later he was deeply comatose with an extradural hæmorrhage. The actor fell less than 6 ft. and it would seem that the rebound was at least one-twelfth of this distance. This rebound is damped down by a number of factors such as the semi-fluid content of the skull, the attachment of the trunk, etc. It is also reduced when the kinetic energy is dissipated in the production of a fracture, as in this instance.

The inbending of the skull which occurs at the moment of impact is then replaced by a transitory

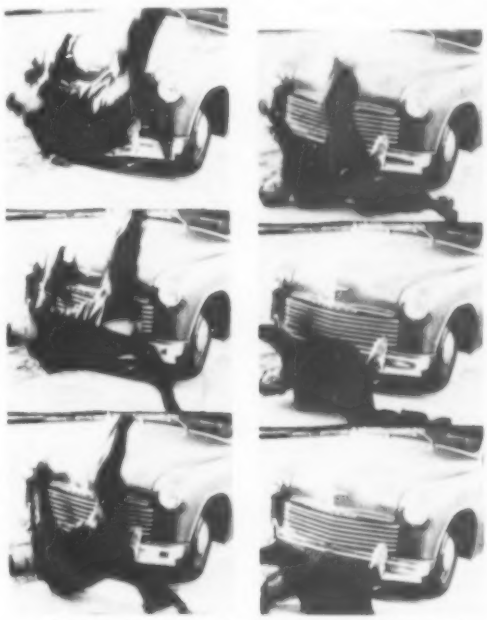


FIG. 82.—Portion of a cine film exposed at 16 frames per second, showing the impact and rebound which resulted in an extradural hæmorrhage.

outbending of the skull in the same region. It can be easily demonstrated by a model that this mechanism will tend to strip off the attachments of the dura within this area, and so a potential extradural space is produced into which the hæmorrhage can take place. This mechanism has one important clinical correlation. The dural separation tends to be centred around the point of impact, and so the resulting extradural clot is most easily located by exploring in relation to the point of impact rather than in relation to the fracture line.

This principle is of importance in the determination of the location of the hæmatoma which is more certainly related to the point of impact than the fracture line. Consider two patients—in the first a blow in the frontal region may cause a fracture line radiating down into the temporal region, as shown by Gurdjian, Webster, and Lissner (1953). The fracture line may cross the middle meningeal vessels below the origin of the posterior branch, and hæmorrhage originates from this point (*Fig.* 83). In the second patient the blow is in the midparietal region and the lines of stress radiate again into the temple, and again the fracture line crosses vessels, causing

hæmorrhage from the same point. From observations made in this series, the extravasation in the first instance will be in the anterior fossa and will be related to the point of impact, while in the second the hæmorrhage will accumulate in the temporal and parietal regions underlying the lateral impact.

It therefore appears probable that a potential extradural space is formed by the mechanical

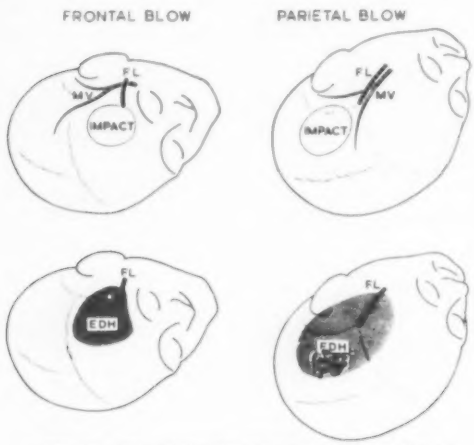


FIG. 83.—The relationships between the site of impact, the fracture line (FL), the meningeal vessels (MV), and the site of the hæmatoma (EDH), indicating the more constant relationship between the site of impact and the hæmatoma than between fracture line and hæmatoma.

deformation of the skull immediately after the impact, and that the initial accumulation of blood-clot is confined to this area. That this area may later be extended seems possible, but it is difficult to accept without reservation the theory put forward by Paul (1955). When the dura is stripped off many branches of the middle meningeal vessels, both arterial and venous, are torn, and bleeding may originate from both sources. It seems probable that the arterial channels, by contraction of their more muscular wall, may be more self-sealing than the veins, and that bleeding due to stripping of the dura *per se* may be predominantly venous in origin. It has been observed that arterial bleeding, when it does occur, usually originates at the point where the fracture line crosses the line of the trunk or major branch of the middle meningeal artery. If the fracture line is wide, blood may extrude through this and appear as a large scalp hæmatoma constituting what Rawling (1904) called a "safety valve", and the leakage to the exterior slows down the intracranial accumulation with a concomitant slowing up of the development of clinical signs. On three occasions comminution of the fracture occurred and a large hæmatoma developed in front of the ear for several days, before the pressure effects of the intracranial hæmatoma were obvious.

There are 2 patients in whom the clinical findings are not satisfactorily explained in this manner.

One was a 35-year-old woman who sustained a mild head injury without skull fracture and a fracture dislocation of the cervical spine in a motor-car accident. A few days later a pair of skull tongs was applied to the skull for skeletal traction. A few hours later she was comatose and when seen at this stage she showed bilateral extensor rigidity, one fixed dilated pupil, and the other pupil rapidly dilating and no longer reacting to light. At operation a large extradural clot was evacuated and a bleeding-point found in relation to the drill hole for the skull tongs.

It is possible that the dura had been separated from the bone at the time of the previous injury, but it must be admitted that the direct accumulation and the raising of the dura by the clot itself seemed more likely in this case.

The other patient was a man of 44 years who sustained an impact in the posterior parietal region and during the next four days developed a typical picture of early extradural hæmorrhage. At operation the fracture was found to pass along a groove which had been made by the healing of a previous fracture.

Why an almost identical injury and fracture should produce an extradural hæmorrhage on the second occasion and not on the first could not be satisfactorily explained by a careful examination of the pathological anatomy at operation, or by subsequent inquiry concerning the method of injury.

EFFECT ON THE BRAIN

As the extradural hæmatoma increases in size the brain is subjected to distortion, local compression, and a generalized increase in intracranial pressure. The effects of the first two factors vary somewhat with the situation of the extravasation. Distortion is probably the cause of headache, and combined with local compression results in the herniation of the medial surface of the temporal lobe down into the tentorial opening, producing further distortion of the brain-stem and, more particularly, of the third cranial nerve. Derangement of the blood-supply of the brain-stem may result in venous obstruction or tension upon the branches of the arteries, as postulated by Johnson and Yates (1956). In unilateral posterior fossa hæmorrhages the distortion of the brain-stem may cause cranial nerve palsies (Hooper, 1954).

Increased intracranial tension is mainly the result of cerebrospinal fluid obstruction at the tentorial level, and produces an acceleration of all these mechanisms which result in compression of the vital centres in the posterior fossa.

CLINICAL FEATURES

The clinical features of extradural hæmorrhage are the result of different combinations of variables: (1) The degree of initial injury and the depth of initial unconsciousness. (2) The site of injury, the position of the hæmorrhage, and the rate of the extravasation. (3) The presence or absence of intradural complications.

But the clinical signs may be discussed as: (1) General features. (2) Features dependent upon the location of the hæmatoma.

The general features are concerned with the immediate effects of the injury and subsequently upon the effects of the expanding lesion on the conscious state of the patient. As already indicated,

the usual head injury is mild and the initial loss of consciousness is not great. In this series of 83 patients 21 did not lose consciousness, and in 24 the initial loss of consciousness was transient and did not exceed 15 minutes in duration. In 16 patients the loss of consciousness was longer, but the patient regained consciousness at some time before operation. Of the remaining patients the initial information was either inadequate or they failed to regain consciousness before operation, which varied between 4 hours and several days after the injury.

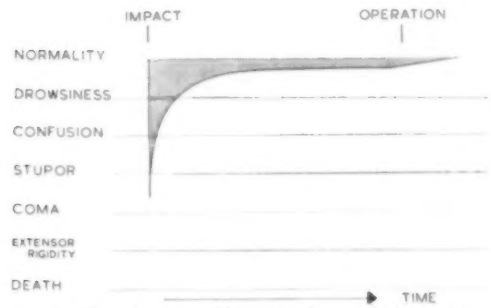


FIG. 84.—Pattern 1. Diagnosis and operation before the hæmorrhage can produce a subsequent deterioration of the conscious state.

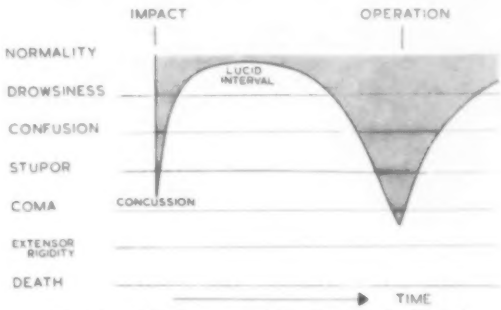


FIG. 85.—Pattern 2. The 'classical' clinical picture in which a lucid interval occurs before the hæmorrhage causes an obvious deterioration in consciousness.

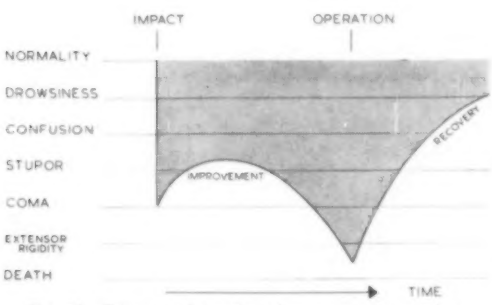


FIG. 86.—Pattern 3. A transitory improvement in consciousness is followed by continued deterioration.

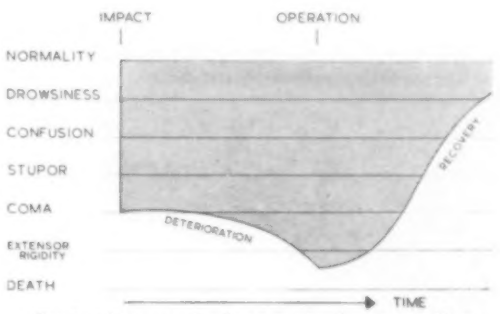


FIG. 87.—Pattern 4. Continued deterioration occurs without evidence of improvement at any stage.

The changes in the conscious state varied with the degree of initial injury and the rate of development of the hæmatoma. Four well-defined patterns are seen:—

1. After an initial period of unconsciousness there was recovery, and operation intervened before any subsequent deterioration took place (*Fig.* 84). There were 8 patients in this group. It is probable that all these patients would have shown the more 'classical' picture if operation had been further delayed.

2. The initial period of unconsciousness is followed by recovery and a 'lucid' interval occurs before expansion of the hæmatoma causes a deterioration in the conscious state (*Fig.* 85). Thirty-seven patients (44 per cent) presented this 'classical' clinical picture. There is little doubt that extradural hæmorrhage is the commonest cause of this sequence after head injury, meningitis, subarachnoid, subdural, and intracerebral hæmorrhage and post-traumatic epilepsy being the only other likely possibilities.

3. Transitory improvement in the conscious state followed by a continued deterioration occurred in 15 patients (18 per cent) (*Fig.* 86).

4. A continued downward trend without any significant improvement in the conscious state was found in 16 patients (19 per cent) (*Fig.* 87). In the remaining patients information concerning the early stages was inadequate.

If operation is not undertaken the conscious state passes through a stage of drowsiness, confusion, stupor, and finally, coma. This last stage was observed in 48 patients in whom there were 16 deaths, and was associated with decerebrate rigidity in 13 (5 deaths), respiratory distress in 22 (10 deaths), hyperthermia in 6 (4 deaths), and abnormal pulse and blood-pressure recordings in 18 patients (10 deaths). Acute pulmonary œdema occurred in 2 cases.

Investigation into the features dependent upon the location of the hæmatoma indicates that there are certain differences in the groups which were distributed as shown in *Fig.* 88.

Group 1: Extradural Hæmorrhages of the Anterior Fossa.—In this series the hæmorrhage was largely or solely confined to the anterior fossa

in 15 patients. The impact was received in the lateral frontal region in all, and it was interesting to observe that two-thirds of the patients were involved in traffic accidents. This high proportion is probably related to the frequency of head-on impacts with injuries of this origin.

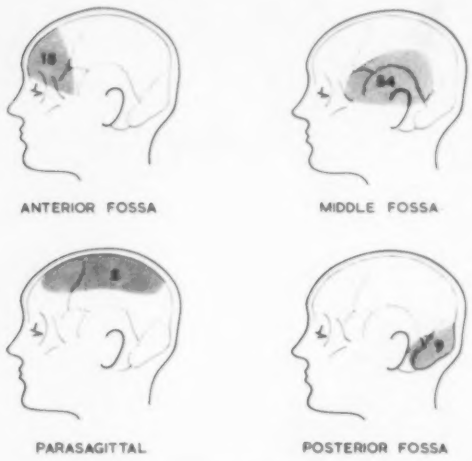


FIG. 88.—The distribution of the extradural hæmatomata.

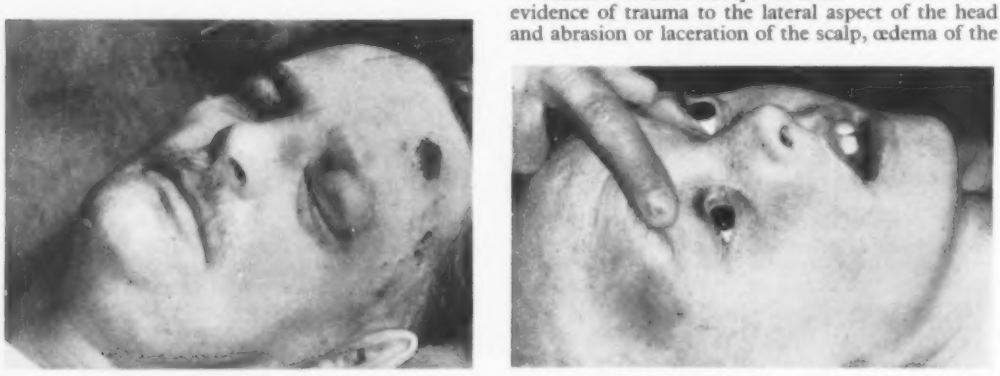
Fractures of the skull occurred in all except one hæmophilic child aged 8 years. The frontal bone alone was involved in 5 patients and the temporal bone alone in 3, while in 5 patients the fracture involved both frontal and temporal bones. In most cases the fracture radiated down across the anterior temporal region, involving the anterior branch of the middle meningeal vessels in 4 patients and the main trunk in 6.

In general, the progress of the clinical picture was much slower than in the middle fossa group, the average interval between injury and operation being 38 hours. Signs of pyramidal involvement were less obvious and tended to appear later. They occurred in 9 patients, but were mild, being limited to inequality of the reflexes in 6, while only 4 exhibited obvious paresis. Extensor rigidity was less marked and again less frequent, appearing in only 4 patients at the time of operation. Pupillary inequality was present in 10 in this group, a figure rather at variance with the proportion which developed extensor rigidity, for it is generally conceded that these two phenomena are related to the same mechanical derangement, namely the herniation of the temporal lobe into the tentorial notch. All patients showed considerable deterioration of the conscious state. At the time of operation 8 were stuporose and 7 were comatose. Three patients died, a mortality of 20 per cent.

The clinical picture of this group may be summarized thus: As a result of a frontal impact, causing orbital hæmatomata and a fracture in the fronto-temporal region, there is a gradual deterioration of the conscious state. Pupillary inequality occurs frequently, but pyramidal signs are not marked and occur late. The diagnosis is usually made after consideration of the nature of the injury and the steady deterioration of consciousness (*Fig.* 89).

Group 2: Extradural Hæmorrhages of the Middle Fossa.—In this group of 54 patients there were 43 histories which were complete enough for detailed analysis. In these it was found that the traffic accident was a much less significant aetiological factor, occurring in only 12 patients (27 per cent), while there were 23 patients (53 per cent) in whom this complication followed falls upon the head. These figures form a significant contrast with those found in anterior fossa hæmorrhages.

Almost without exception there was some local evidence of trauma to the lateral aspect of the head and abrasion or laceration of the scalp, œdema of the temporal fossa (*Fig.* 90), or a fissure fracture observed in the radiograph.



A B

FIG. 89.—A, An anterior fossa hæmorrhage producing stupor 4 days after injury. B, An anterior fossa hæmorrhage in a hæmophilic child producing deep coma and pupillary dilatation.

The development of the clinical picture was variable. In 16 patients there was no initial loss of consciousness, it was transient in 13, while in 8 patients consciousness was recovered before operation. In 6 patients operation was carried out although the patient was not deteriorating further, because the clinical signs strongly suggested an extradural

hæmorrhage. Twenty-two patients had a 'classical' lucid interval, while 7 showed some degree of recovery before markedly deteriorating; 10 showed progressive deterioration over the period of observation (*Fig.* 91 A, B).

There was a very considerable variation in the rate of development of the syndrome, even in the so-called 'classical' cases, but it seems characteristic of the middle fossa hæmatoma that the rate of development is greater than in other situations, for 40 per cent in this group required operation within 6 hours of injury, while a total of 64 per cent required operation within 12 hours. There was, however, a significant number of patients in whom the hæmorrhage appeared to have ceased, and the increase in the size of the clot which necessitated operation was probably due to osmotic factors in the same manner as is seen in the subdural clot. In 4 patients operation was not performed until after the tenth day. In one of these there was cerebrospinal fluid leakage from the ear which masked the presence of clot for 10 days. A similar finding has been observed by Connolly (1956).

Pyramidal signs, contralateral weakness of the face, hand, and, less obviously, the leg, were present in 50 per cent of the patients, while pupillary dilatation on the affected side was slightly more constant (*Fig.* 92). In the more rapidly developing cases the stage of hemiparesis was often missed or not observed and the patients passed directly into a state of decerebrate rigidity. A significant proportion (40 per cent) in this group also sustained intradural hæmorrhage, and the overall mortality was 25 per cent.

Summarizing these findings, it is seen that the middle fossa collection is the most common. It is more frequent after falls or blows upon the head than after traffic accidents. There is clearly defined evidence of a blow on the lateral side of the head.

There is great variability in the clinical picture presented, but it may be said to pass through five stages (*Fig.* 93):—

1. Brain displacement, during which the patient complains of severe headache.

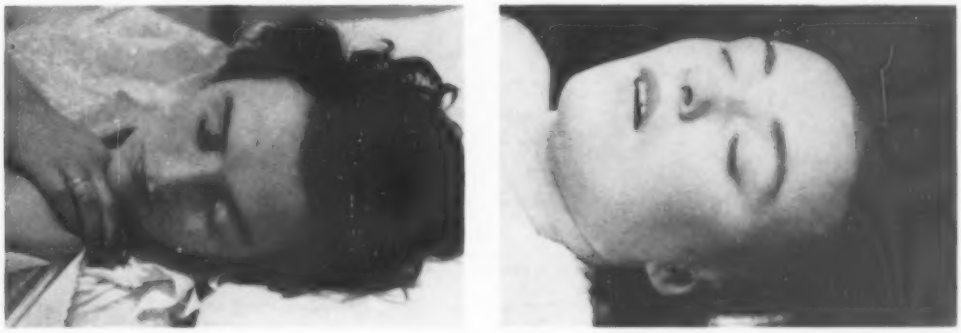
2. Local compression, producing weakness of the contralateral face and limbs.

3. Temporal lobe herniation; the displacement of the medial side of the temporal lobe medially and downwards over the free edge of the tentorium produces a paralysis of oculomotor nerve function—first a pupillary dilatation and later paralysis of the recti.

4. Brain-stem disorders consequent upon vascular changes induced by the temporal lobe herniation and dislocation of the brain-stem. These changes induce changes in the state of consciousness, extensor rigidity, and paralysis of ocular muscles.

5. The final stage—medullary compression. Respirations become irregular and cease, cardiac arrest follows later. In some rapidly developing extradural hæmorrhages in young subjects acute pulmonary œdema occurs in this final phase.

FIG. 90.—The scalp contusion and œdema of the temporal fossa which became obvious after shaving the scalp.



A B

FIG. 91.—A, Photograph obtained soon after admission shows a drowsy patient. B, Thirty minutes later the patient is in coma.

Group 3: The Parasagittal Hæmatoma.—A hæmatoma occurred in the parasagittal region five times. There was no distinct relationship between the nature of the accident and the occurrence of a hæmorrhage in this situation, but it was observed that there was always some local sign of injury in the parasagittal region. The rate of development of the clinical syndrome was very variable, but pyramidal signs and pupillary inequality appeared more constantly, being present in 4 of the 5 cases encountered. No patients in this group died.

Group 4: The Posterior Fossa Hæmatoma.—There were 9 patients in whom the hæmatoma

originated in the posterior fossa and most of these have been previously described (Hooper, 1954). The type of accident causing this hæmatoma was not very specific, but it is noted that it followed traffic accidents on 5 occasions. The injury varied in severity, but it was always directed to the posterior half of the head, and the site of the impact was apparent in all cases, and a fracture crossing the line of a venous sinus was present in all except one patient. The occurrence of a lucid interval corresponded with that found amongst extradural hæmorrhages generally.

FIG. 92.—A late stage in the development of a middle fossa hæmorrhage. The pupil is dilated and fixed, and pulmonary œdema is developing.

The progression of the clinical syndrome varied, and acute, subacute, and chronic persistent forms have been described. In the acute forms consciousness may be lost abruptly and soon followed by pupillary dilatation and signs of medullary compression, while in the delayed or chronic persistent form consciousness is usually retained.

Neck stiffness, headache, and vomiting were fairly constantly present, while cerebellar signs and cranial nerve palsy indicated local distortion of the cerebellum or brain-stem. Papilloedema occurred only in the delayed forms. The mortality was highest in this group, in which 4 of the 9 patients died. These fatalities were confined to acute cases and occurred in the earlier years covered by the series, before the significance of the clinical picture was fully appreciated.

BRAIN-STEM LESIONS

The impaction of the uncinate gyrus in the tentorial notch distorts the cerebral peduncles and causes gross derangement of the vascular mechanisms therein. These disturbances have been attributed to venous stasis by Scheinker and Evans (1945) and others, while Johnson and Yates (1956) suggest that the distortion causes rupture of the small arterioles within the brain-stem.

Macroscopically, hæmorrhages may be seen following the line of the vessels and these may coalesce to form larger extravasations.

Associated with these hæmorrhages it is possible to demonstrate œdema, liquefaction, and later macrophage absorption in areas surrounding or related to these hæmorrhages. At a much later stage gliotic scarring can be seen (*Fig.* 94 A, B, C).

The vascular changes have a characteristic distribution, occurring most frequently in the tegmentum of the midbrain lying in front of and lateral to the central column of grey matter, i.e., in the medial and lateral areas of the reticular formation and in relation to the red nucleus and the long tracts in its proximity (*Fig.* 95). The nuclei of the ocular nerves may also be involved. The hæmorrhages are less frequent in the pons, where they may occur in the lateral portion of the tegmentum medial to the vestibular nuclei.

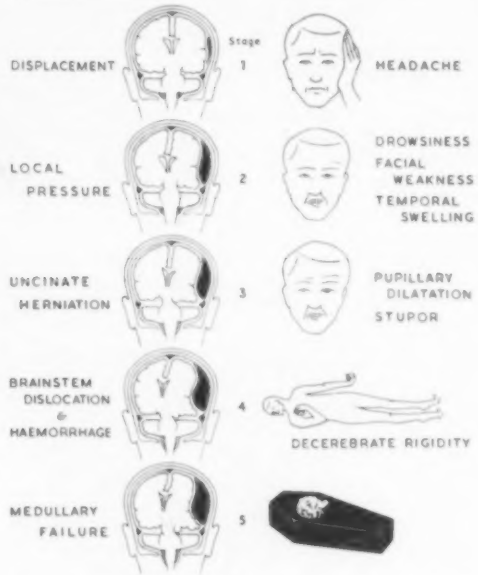


FIG. 93.—Stages in the development of the clinical picture in middle fossa hæmorrhage.

These vascular derangements are responsible for: (1) Changes in the state of consciousness; (2) disturbances in muscle tone; (3) paralysis of ocular muscles; (4) disturbances of the long tracts producing (*a*) gross disintegration of movement, and (*b*) disturbance of balance.

In discussing the state of consciousness in midbrain lesions, Cairns (1952) describes four forms: (1) Coma with decerebrate rigidity. (2) Hypersomnia. (3) Akinetic mutism. (4) Coma with hyperthermia.

Among patients with extradural hæmorrhages all may occur, but coma with decerebrate rigidity is that most frequently seen in the acute and preoperative stage, being found in 13 patients. Wide variations occur in the intensity of this state, which may range from a mild increase in extensor tone associated with light coma to the deeply comatose patient with intense extensor rigidity of all limbs and trunk (*Fig.* 96), with frequent tonic fits and paroxysmal hyperpnœa which may be induced by light sensory stimuli, a phenomenon which seems out of keeping with the depth of coma observed.

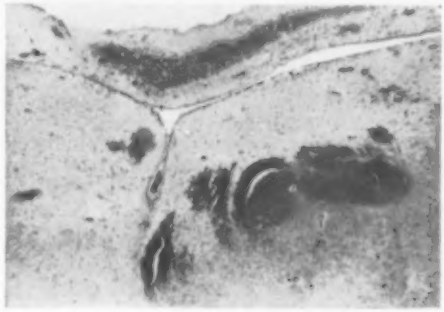
Hyperthermia is usually associated with this decerebrate state, but often the hyperthermic patient

is found to be deeply comatose, flaccid, with diminished or absent reflexes, a rapid pulse-rate, rapid shallow respirations, and the skin is often cold and gives no indication of the high internal temperature. In the later stages dehydration occurs and is followed by peripheral circulatory failure.

These states of coma are frequently seen before operation in advanced cases of extradural hæmorrhage and may persist after operation for varying times, until either the death of the patient or some degree of recovery occurs. In the latter event the state of the patient gradually changes into one of hypersomnia or akinetic mutism.

Some degree of recovery may occur in these patients, but occasionally a chronic and persistent state continues, from which the patient is only released by death from intercurrent infection or other extracranial influences. These changes may be associated with phenomena which are more closely related to hypothalamic disorders—persistent temperature instability, abnormal sweating (especially on the face), and emotional changes may be seen in the recovery phase. Disturbances of oculomotor nerve movement due to local involvement of the nuclei and connexions in the central grey matter are frequent. Varying degrees of isolated paralyses may occur together with abnormal pupillary reactions. Frequently there are gross defects in co-ordination of eye movement. As recovery occurs the disturbances of motor function may become more obvious. There may be a spastic hemiparesis or a unilateral loss of co-ordination. It would appear that an increase of tone on one side accompanied by incoordination of movement on the other is a frequent sequel of this midbrain disturbance. The equilibrium is grossly disturbed and it may be many months before the patient can support himself. Speech is usually affected and often assumes a staccato form.

The occurrence of these midbrain phenomena has a profound effect on prognosis. The mortality, even after immediate operation, is high; the post-operative period is stormy; convalescence is protracted and recovery frequently incomplete. Of 14 patients who exhibited such changes 8 died, while 2 of the survivors showed persistent changes in tone and co-ordination of voluntary movement.



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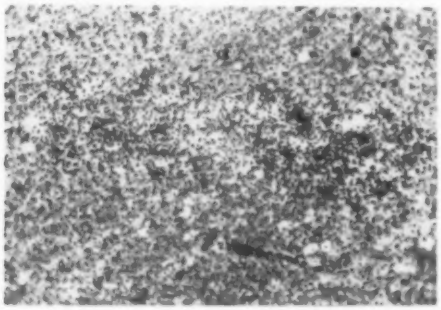

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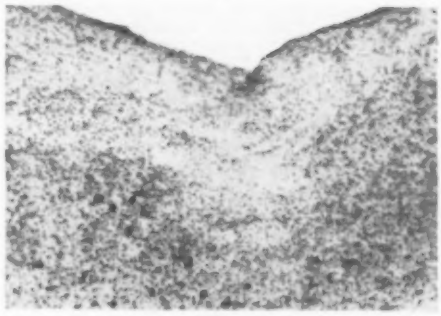

C

FIG. 94.—Photomicrographs. A, Midbrain hæmorrhages situated anterior to the aqueduct of Sylvius. (× 18.) B, Gitterzellen appearing in an area of necrosis, the patient dying 3 months after injury. (× 44.) C, A central area of gliosis lying in front of the aqueduct in a patient who died 6 months after injury. (× 44.)

FIG. 95.—Extensive area of necrosis (cross-hatched) extending from brain-stem to hypothalamic area. This patient sustained a respiratory arrest before operation and remained in a decerebrate state until his death 3 months later.

FIG. 96—Extensor rigidity occurring before operation and persisting in the post-operative period.

OTHER INTRACRANIAL LESIONS ASSOCIATED WITH EXTRADURAL HÆMORRHAGE

In the autopsy series reported by LeCount and Apfelbach (1920) of 104 supratentorial extradural hæmorrhages large enough to produce compression, only 6 had large subdural clots, 25 had smaller amounts (less than 20 ml.), while in the remainder the brain injury was trivial, and in 15 extradural hæmorrhages in the posterior fossa all had some degree of cerebral contusion. The coexistence of other intradural lesions has often been blamed for the high mortality of this condition, so it is important to assess the frequency and nature of these lesions and their general relation to the mortality associated with extradural hæmorrhage.

It is certain that intradural lesions are not uncommon in the presence of extradural hæmorrhage. Gurdjian and Webster (1942), in 30 cases of extradural hæmorrhage of middle meningeal origin, found subarachnoid hæmorrhage in all patients in whom a lumbar puncture was performed (about 50 per cent). Significant intradural lesions were found as follows: subdural hæmatoma, 7 cases; temporal lobe clot, 3 cases; subdural effusion, 2 cases.

Goinard and Descuns (1948), in 43 cases, found subdural hæmatomata in 11, intracerebral clot in 3, and subdural effusion in 4 patients. Sartorius and Humphries (1946) found an associated cerebral injury in 13 of their 20 patients. In the present series, subarachnoid hæmorrhage was confirmed 15 times. Subdural hæmatomata or effusions were found on 27 occasions and only 9 of these were large; 6 were small, being less than 0·1–1 cm. in thickness. They occurred 20 times on the same side as the extradural hæmorrhage and 7 times on the opposite side. Intracerebral hæmatomata were found 15 times and these were usually associated with subdural hæmorrhages. They occurred on the same side as the extradural hæmorrhage in 13 patients and on the opposite side in 2. It is interesting to assess the degree of injury associated with the appearance of these complications. Omitting the subarachnoid hæmorrhage we find 32 patients with intradural hæmorrhage in association with extradural hæmorrhage. In estimating the degree of initial unconsciousness it was found that in 8 patients insufficient information was available over the early period, but 5 had no loss of consciousness while 3 others had only a transient loss. Thirteen had fair recovery of consciousness before operation, while in 3 only was the initial state of unconsciousness prolonged until operation intervened. It was possible to classify the degree of injury from the degree of fracture present and the duration of unconsciousness, and assessed in this way one finds: injuries of mild severity, 13 patients; moderate severity, 12 patients; severe degree, 5 patients; not classified, 2 patients. Thus the injuries sustained by the patients in this group were not significantly more serious than the rest of the series. Twenty-three of the 32 patients were talking at some stage between injury and operation, the most striking instance being that of a man of 65 years who fell from the tray of a truck and was unconscious for 5 minutes, but in 7 hours developed a large extradural hæmorrhage and a cortical laceration with an underlying extensive intracerebral clot. Another young man who fell from a bicycle was not initially unconscious, but 3 hours later there was a cessation of respirations due to a combination of a posterior fossa extradural hæmorrhage and a subdural hæmorrhage on the opposite side. Seven patients were admitted comatose and only 3 of these had remained in coma since the initial injury; 4 showed varying degrees of drowsiness or confusion.

The presence of these complications, severe though some of them were, was not necessarily associated with severe injuries, in fact, most corresponded well enough to the classification of 'not of severe degree', though often said to be characteristic of the injury causing extradural hæmorrhage. Thus we find that in any series of extradural hæmorrhage intradural clot is likely to be found in 30 per cent of patients, but there is little indication from the nature of the injury which cases are liable to develop such a complication.

The mortality associated with intradural clots is about 48 per cent in this series, which is four times higher than in the uncomplicated form (12·5 per cent).

It seems probable that the mortality in this group can be significantly reduced. Most of the deaths occurred in the early years covered by the survey, and these cases might have survived if the present routine of looking for intradural complications at the time of the initial operation had always been observed.

RADIOTHERAPY

Early radiography is an important factor in the early diagnosis and ultimate survival of patients with extradural hæmorrhage. In some instances the knowledge that a fracture is present helps to establish that the deterioration of the conscious state is traumatic in origin. The situation of the fracture allows one to predict the site of impact, and its relation to the meningeal vessels indicates the possibility of an extradural extravasation. Operative evidence indicates that in almost all cases the origin of the major portion of the bleeding comes from the meningeal vessel where it is crossed by the fracture line.

Radiographs may indicate that some accumulation of clot has already occurred when a displacement of the pineal shadow can be identified, as in the cases reported by Falconer and Schiller (1942) and Madonick and Oljenick (1945). In 3 patients in this series a shift of the pineal shadow confirmed the provisional diagnosis.

Angiography has a limited use in most extradural hæmorrhages for the diagnosis can usually be made without this procedure, and in most cases the need for early operation contra-indicates an investigation which will delay the patient's transfer to the operating theatre. Moreover, there may be difficulties in distinguishing the extradural hæmatoma from the subdural, although Norman (1956) indicates some points by which a distinction may be made. In the more delayed types and in those in which the history was obscure, this investigation was of importance and was utilized on 3 occasions. In one of these patients, previously reported by Jamieson (1952), the angiogram indicated the situation of the tear in the middle meningeal artery by a local extravascular extravasation of contrast medium.

Air studies are less frequently used, but may yield vital information. They were essential for the diagnosis of a posterior fossa collection when no fracture was present. In another patient, a temporal extradural collection was suspected, but was not found when an exploratory burr-hole was made in this region. The lateral ventricle was then needled through this opening and a small amount of air introduced. Radiographs obtained with a portable

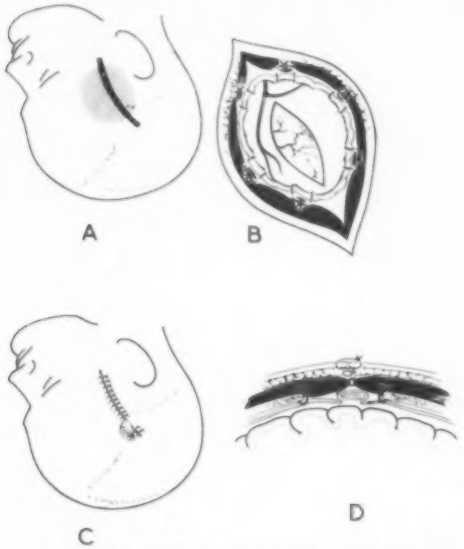

FIG. 97.—Details of operative technique for extradural hæmorrhages of the middle fossa. A, Incision and area of bone removal; B, Dural opening and suspension sutures; C, Position of the rubber drain and sutures; D, The completed operation.

unit in the theatre showed a displacement and deformity of the frontal horn. Within a few minutes the exposure was extended and a large frontal hæmatoma evacuated.

Although radiography can aid diagnosis and facilitate operation, when the patient presents the 'classical' signs or shows evidence of respiratory depression, operative intervention must not be delayed by radiographic procedures.

OPERATIVE TECHNIQUE

There are a number of important points in the management of extradural hæmorrhage (*Fig.* 97).

1. Initial Exploration.—The site chosen is related to either the site of impact, if this is known from the history, to signs of trauma on the scalp after it has been shaved, or to the fracture line seen in the radiographs of the skull.

2. The Bone Opening.—Inadequate access does not allow complete hæmostasis or complete removal of the clot. A ridge of clot may be left around the periphery of the cavity which promotes the formation of further clot, and thereby prevents complete expansion of the brain. The bone opening may need to extend beyond that indicated in the classical subtemporal operation. If facilities for making an osteoplastic or free bone-flap are not available, a large bone opening may be obtained by nibbling, and the resulting bone defect dealt with at a later stage (*Fig.* 98).

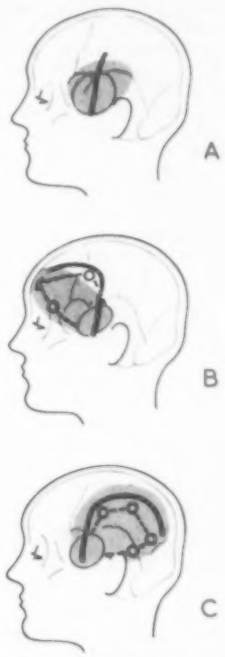

FIG. 98.—Diagrams indicating the extension of the incision to allow the elevation of an osteoplastic flap, for access to hæmatomata in the anterior fossa (B) and the posterior parietal and parasagittal regions (C).

3. Evacuation of the Clot and Hæmostasis are problems which, although they may produce difficulties, do not give rise to any special need for discussion.

4. Intradural Inspection.—The dura should always be opened and the brain and subdural space observed for collections of clot or fluid so that these conditions may be removed as soon as possible. The incision in the dura also aids in the completion of the next stage.

5. Obliteration of the Dead Space.—The cavity remaining after removal of the clot must be obliterated in order to prevent re-collection of clot. The expansion of the brain after removal may take place rapidly and no further procedures may be necessary, although it is wise to take precautions against clot re-forming. In most cases the cavity can be adequately obliterated by suturing the dura to the muscle or pericranium around the periphery of the bone opening, and this procedure is facilitated by first opening the dura. Should the brain itself still fail to expand, a lumbar puncture needle should be placed in the lumbar subarachnoid space and sterile Ringer's solution or normal saline injected until the cerebral hemispheres are pushed up and out of the tentorial opening and the ventricles distended.

If there is no laceration of the arachnoid membrane the brain will distend completely to fill the intracranial cavity. On occasions the comatose patient has suddenly awakened during this procedure. The injection of Ringer's solution may be made into the lateral ventricles, but often these are so distorted by compression that such a procedure is difficult and not so easily controlled as a lumbar subarachnoid injection. This procedure has considerably reduced the cases in which it has seemed advisable to incise the tentorium because of the fear that an unreduced tentorial herniation has maintained a state of coma. This procedure has been advocated in other types of intracranial hæmatomata and has also been advocated by LaLonde and Gardner (1948); Goinard and Descuns (1948) advocate the inflation of the ventricles by a brain needle introduced through a separate burr-hole, but it has seemed easier to turn the patient on his side and introduce a lumbar puncture needle into the lumbar subarachnoid space. It seems probable that the injection of saline in this way helps to push any downward herniation of the temporal lobe out of the tentorial notch. For this reason, whenever possible, the operation commences with the patient lying on his side. This position not only helps to maintain an effective airway but allows easy access to the lumbar spine. It is felt that this procedure alone has saved lives since it was adopted as a routine four years ago, and this view is supported by Gillingham (1954) and Robinson (1958).

6. The Insertion of a Drain down to the level of the dura and the closure of the wound in anatomical layers complete the operative procedure.

Adequate resuscitation is so important that on the discovery of an extradural hæmorrhage preparations for an intravenous infusion and a blood transfusion must be initiated without delay.

No less important is the post-operative management. Resuscitation must be continued until the blood-pressure is stable at a satisfactory level. Care must be taken to maintain an adequate airway and oxygenation. If this cannot be maintained by posturing, a tracheotomy should be performed. The appearance of hyperthermia usually signifies damage to the brain-stem but no longer indicates an early and fatal termination. Early and energetic measures for temperature control have made deaths from this complication rare.

RESULTS OF OPERATION

In this series of 83 patients, there were 19 deaths (23 per cent). One patient died before operation, while 3 deaths occurred much later, and, in fact, in patients in whom recovery from the effects of the injury were not sufficiently complete for them to carry on an independent existence.

CAUSES OF DEATH

The causes of death in the 19 patients are set out in *Table V*.

In 4 of the 6 patients who suffered respiratory failure spontaneous respirations occurred after removal of the clot, but the patients failed to respond and subsequently died from pulmonary œdema, hyperthermia, or other complications. It is interesting to note that rapidity of onset or massive intradural hæmorrhage caused death on only 4 occasions, while exsanguination following rapid removal of a large clot causing severe compression produced cardiac arrest in 2 patients before blood could be typed for transfusion.

Late chest complications accounted for 3 patients at varying periods between 3 days and 3 months after injury. One old lady probably died from a coronary occlusion two months after injury in which she sustained brain-stem damage and from which she had made a slow and imperfect recovery.

Table V.—Causes of Death in 19 Patients

Respiratory failure before operation	6
Rapidity of onset	1
Massive intradural injury	3
Exsanguination	2
Inadequate removal	1
Recurrence	1
Failure to locate clot	1
Chest complications	3
Old age (? coronary occlusion)	1

Behind all these causes of death lies the major factor—delay in operative intervention. In this series 13 of the 19 deaths could well be attributed to delay in operative intervention. This delay occurred before admission to hospital on 4 occasions, while in 10 patients the delay occurred after admission while the patient was under observation. In 9 of these 13 patients the clinical picture was reasonably clearly that of an expanding intracranial lesion and in 4 of these the picture might be considered to correspond to the 'classical' description of extradural hæmatoma.

FACTORS CONCERNED IN SURVIVAL

1. Nature of the Injury.—In 1886 Jacobson, in his grouping of extradural hæmorrhages, set a standard which has been accepted ever since. He divided the cases in his series into three groups: (1) Most hopeful for trephining—no focal injury to the brain. (2) Less hopeful cases—the fracture involves the base of the skull, brain injuries trivial. (3) Cases probably hopeless from the first—violence very great, extensive fracture, and severe injury to the brain.

A careful evaluation of the information available does not suggest that the mortality is proportional to the severity of the injury. In the present series there is no true correlation between the severity of the impact and death due to intracranial hæmorrhage. As previously stated, this finding is probably related to the nature of the injury and the limitation of the extradural hæmorrhage by brain swelling and intradural hæmorrhage.

Table VI.—Influence of Severity of Injury on Death or Survival

Degree of Initial Unconsciousness	Degree of Injury	Deaths No.	Deaths Per cent	Survivals No.	Survivals Per cent
None	Slight	4	22	19	29
Transient	Mild	6	33	18	28
Moderate—recovery before operation	Moderate	3	17	14	21·5
Unconscious until operation	Severe	5	28	14	21·5

When the deaths in this series are analysed, it is found that the severity of the injury, as judged by

the initial depth of unconsciousness, is in most cases relatively mild. Of the 19 deaths, 4 had no initial loss of consciousness, 6 had only transient loss, 3 recovered consciousness before operation, and only 6 remained unconscious until operated upon. *Table VI* shows that the same proportions are seen in the survivals.

It may be said that in severe injuries the extradural hæmorrhage is often not detected because the patient dies rapidly, but it is probable that the intradural hæmorrhage and swelling not only kill the patient before any significant collection can occur in the extradural plane, but also limit the rate at which extravasation can occur. Sometimes the nature of the injury bears a definite relationship to the balance between death and survival. In the series reported by Sartorius and Humphries (1946) from South Africa, we find only 1 death in 20 patients. These authors attribute the very high survival-rate to the fact that the injuries occurred amongst South African natives who had beaten each other with sticks, thereby producing localized types of head injury.

2. Rate of Bleeding.—This factor may be significant for it is found that among the 8 patients who were operated upon within 6 hours, 2 died. On the other hand, if reference is made again to the last 30 consecutive cases, no clear conclusion can be drawn, because only 1 death occurred among patients with arterial bleeding and 1 with mixed arterial and venous bleeding. In the first case the patient was old and died some months after operation, and in the second case death was due to cardiac arrest in the later stages of the operation.

3. Intradural Complications.—Of the group of 32 patients who developed localized intradural lesions in association with the extradural hæmorrhage, 11 died. Among these 11 deaths we find only 3 massive hæmorrhages. At least 4 deaths were due to tardy recognition of the extradural hæmorrhage.

Although it is admitted that intradural complications diminish the chance of survival, their importance in this respect should not be over-emphasized. Thirteen of the 20 cases reported by Sartorius and Humphries sustained intradural hæmorrhage, but only 1 death occurred in this group. Thus there is evidence that if looked for and treated the outlook is not statistically much worse than in the uncomplicated groups.

4. State of Consciousness at Operation.—The figures provided by Munro and Maltby (1941) comment upon the relationship between deterioration in the conscious state and the mortality-rate. Of those in deep stupor or coma, 71 per cent died; of those who were drowsy or verging on the state of stupor, 46 per cent died; and of those who were conscious at the time of operation, 17 per cent died. In the present series the relation to the state of consciousness is also well seen, for deaths only occurred frequently when coma was established (*Fig.* 99). No deaths occurred among the group of patients who were either conscious and rational or only confused at the time of operation. Of the 17 patients in a stuporose condition there were 2 deaths, while of the 48 patients who were operated upon in a comatose condition, 17 died. When the state of coma was complicated by decerebrate rigidity, as it was on 13 occasions, 5 of these patients died, while of the 18 who exhibited respiratory distress, 10 died (*Table VII*).

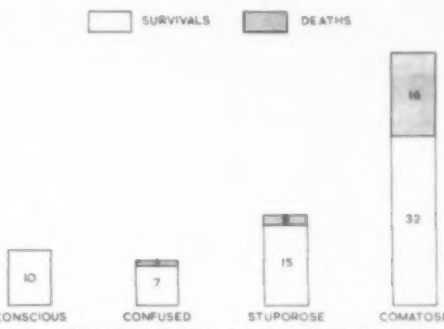

FIG. 99.—The relation between the state of consciousness at the time of operation and the mortality.

5. Age.—From this series it might be difficult to draw any conclusions about mortality in relation to age because the extreme ends of the age-scale contain only a handful of cases, but it seems likely that the injury is not well tolerated in the older groups (*Fig.* 100). Four out of 6 patients over the age of 60 years died, while in children only 1 of the 14 children under 10 years and 2 of the 12 children in their teens died. Campbell and Cohen (1951), who report only 2 deaths in their series of 20 cases, also assert that the mortality in children is low. There are many factors which may be operating in this group. The absence of alcohol as a confusing factor and the greater care taken in looking after these young lives are probably significant.

Table VII.—INFLUENCE OF THE STATE OF CONSCIOUSNESS ON DEATH AND SURVIVAL

STATE OF CONSCIOUSNESS AT OPERATION	TOTAL		DEATHS		SURVIVALS	
	No.	Per cent	No.	Per cent	No.	Per cent
Conscious and rational	10	12	0	0	10	100
Confusion	8	10	1	12	7	88
Stupor	17	20	2	12	15	88
Coma	48	58	16	34	32	66
Uncomplicated	17	20	3	18	14	82
Extensor rigidity	13	16	5	39	8	61
Respiratory distress	22	27	12	55	10	45

6. Experience.—One of the important findings in the examination of the figures indicating the survival-rate (*Fig.* 101) is the improvement in the later years. In the first half of the period under review, 14 deaths occurred in 34 patients, while in the second half 4 deaths occurred in 49 patients, a mortality of 9 per cent. This very significant fall in the mortality-rate is due in part to increased experience, to improvements in the organization in the hospitals concerned, and in very large measure to an increased awareness of the condition amongst house-surgeons and general practitioners.

The more liberal introduction of the principle of operating on suspicion (Hooper, 1949) rather than awaiting the development of the classical syndrome

has also been a most important factor. Jamieson emphasizes this point and his series of 12 patients with 1 death makes it clear. On the technical side, the suspension of the dura to the pericranium or muscle and inflation of the brain by an injection of saline have diminished recurrent hæmatomata which were frequent causes of failure to respond to operative intervention. The routine search for related intradural complications has also helped to lower post-operative mortality and morbidity.

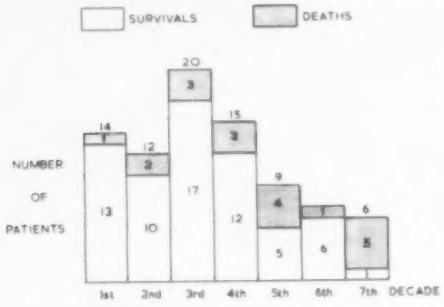

FIG. 100.—The relation between age and mortality.

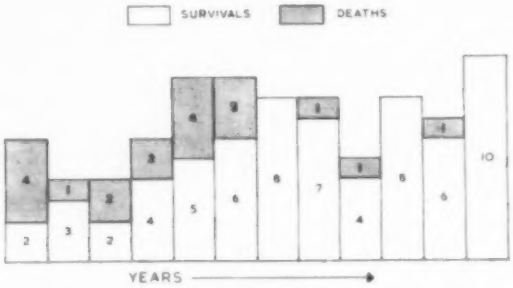

FIG. 101.—The influence of experience upon death and survival.

7. Organization.—In many large general hospitals there is now a tendency to place all the complicated head injuries directly under the care of the neurosurgeon, and those being trained in general surgery or those destined to enter general practice have little experience in diagnosis and less in operative treatment of the acute emergency presented by an extradural hæmorrhage. Brodin (1951) states that in Sweden head injuries remain the province of the general surgeons and claims that they are able to achieve just as satisfactory results as the neurosurgeons, but the analysis of the figures he quotes is not so reassuring. One-third of the patients died without operation, and of the remainder 3 were operated upon after periods longer than 7 days. Thirteen had intradural lesions and 9 of these died. Brodin asserts that the chief problem is not that of organization or therapy but one of diagnosis, and with this fact there can be no disagreement. Nevertheless, organization and management are also important. Munro (1952) states that "these patients should be hospitalized for treatment in large, well-staffed, properly equipped hospitals that have a blood bank or its equivalent available. They can, and should be, moved for any reasonable distance by train, plane or ambulance to conform with this requirement." It is probably insufficiently realized that the operative management of an extradural hæmorrhage seen at an early stage is an easy operation, well within the capabilities of a reasonably trained surgeon, but later cases which have developed the classical syndrome, or have developed involvement of the brain-stem, or have additional intradural hæmorrhages, present formidable difficulties for the surgeon without training in neurosurgical operating technique or post-operative management.

Lewin (1954) discusses the need for transferring complicated head injuries to neurosurgical centres and stresses the need for some neurosurgical training for some general surgeons.

In this country (Australia), where the relatively sparsely populated agricultural communities lie at some considerable distance from the larger cities, the need for some organization occurs. Some conception of the difficulties is illustrated by the following case history. An 8-year-old girl struck her head when she fell from a bicycle, but was able to walk home. Five hours later she became comatose. She was taken to the local 10-bed hospital where her local doctor made an exploratory burr-hole and found an extradural hæmorrhage which he felt he could not control. Arrangements were made for her transfer to the Royal Children's Hospital 129 miles away. She reached this hospital two hours later where the theatre staff were in readiness. At this stage she had bilateral fixed dilated pupils and extensor rigidity. Large extradural and subdural clots were removed and the child made a complete recovery after a few initial stormy days.

Raney, Raney, and Peterson (1953) have suggested that in these circumstances the external carotid artery should be tied in the neck and the patient transported afterwards to a neurosurgical centre. For the occasional operator, ligation of the external carotid artery is no easy task, and it is felt that the undue delay may be prejudicial. Coates (1957) has described a similar case in which partial removal of the clot was carried out and the patient then transferred to the nearest general hospital for completion of the operation. An early recovery was obtained.

On another occasion, the patient had been admitted to a well-equipped provincial hospital, but there were difficulties in the diagnosis of the nature and location of the lesion. A child was struck on the side of the head by a ball. He went home, but later vomited and became drowsy. When seen by his local doctor he was stuporose, with a left fixed dilated pupil. He was taken to a well-equipped provincial hospital 190 miles away from a neurosurgical centre. When seen here his condition had improved. Radiographs revealed no fracture, but he remained stuporose, with a dilated pupil. A telephone conversation then took place between the local surgeon and the author. More details of the actual injury were obtained and an exploratory burr-hole advised at the point of impact (in the posterior parietal region).

A large extradural hæmorrhage arising from the transverse sinus was evacuated and a rapid recovery obtained.

Although it cannot be expected that such happy results will always occur, co-operation between the general practitioner, general surgeon, and neurosurgeon will do much to aid both diagnosis and management. The organization, therefore, should include the training of general surgeons in acute head injuries so that adequate staff and equipment can be maintained at regional or rural hospitals, while rapid transport services should be available to take other patients to more highly organized neurosurgical centres in the cities. Within the hospitals, facilities to deal with these emergencies have improved greatly over the period in which these patients were reviewed. Urgent cases are transferred directly from the casualty department to the operating theatre. Here, improvements in the setting out of the theatre and the use of a sterile emergency set of neurosurgical instruments allows the theatre to be set up within 5 minutes. This last factor has been a major one in reducing the mortality and morbidity in the later cases.

CONCLUSIONS

The following conclusions may be drawn:—

1. The extradural hæmorrhage is usually associated with an injury which, by modern standards, is relatively mild.

2. The impact is received on the lateral aspect of the head with the supratentorial clots and in the occipital region with the less common posterior fossa hæmorrhages.

3. No age is immune.

4. There is great similarity in the basic clinical picture presented.

5. In 25 per cent of cases there is an associated intradural lesion and in less than 4 per cent the condition is bilateral. These complications, if adequately treated, have little influence upon the outcome.

6. The mortality-rate of this condition appears unaffected by the nature of the injury. It is increased in patients over 60 years of age. Intracranial factors which increase the mortality-rate are rapid development of the clot and the presence of intradural bleeding. The major factor influencing mortality is the deterioration of consciousness which may have been reached before operation. This factor is related to delay in diagnosis and treatment.

7. A mortality-rate of 10 per cent should be regarded as a reasonable figure in centres where neurosurgical facilities exist. A higher rate may have to be accepted in other situations, but at the present time a mortality-rate in excess of 28 per cent indicates a low standard of medical education or a poor organization.

Acknowledgements.—I am indebted to the staffs of the Royal Melbourne Hospital and Royal Children's Hospital for their co-operation in the organization and management of the patients included in this series. I would also like to express my thanks to Miss M. Turnbull for secretarial assistance, and to Mr. R. Inglis and Mr. E. Thake for assistance with the illustrations and to Dr. J. D. Hicks for the photomicrographs.

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EXPERIMENTAL SURGERY

SOMATROPHIC PITUITARY HORMONE AND THE LATENT INTERVAL IN WOUND HEALING

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SINCE control of wound infection became a reality, the search for agents which actively speed the rate of wound healing has been widespread. Investigations of healing processes were, in the earlier stages, principally morphological, but more recently biochemical methods such as those used by Dunphy and Udapa (1955) have been more generally adopted. In assessing the effect of different factors on healing, two principal methods have been used. The first is the measurement of the rate of wound contraction and the second the rate of increase of tensile strength. The former, introduced by Carrel (1910), while experimentally convenient, does not entirely satisfy the needs for estimating practical application in surgery (Grillo, Watts, and Gross, 1958). Preferable is the tensile strength method originated by Paget (1853), and fully developed by Howes, Sooy, and Harvey (1929).

In a healing wound it is found that comparison of tensile strength and time produces a sigma-shaped curve. The sharply rising part of this coincides with collagen deposition which, once begun in a wound, proceeds rapidly, the tensile strength so produced keeping pace. Before this phase, however, there is a latent interval, usually of several days' duration. The most efficient method of accelerating healing would, therefore, be one which shortened the latent interval. Changes increasing the amount of granulation tissue would only result eventually in a weaker scar, since it would contain a larger block of collagen. To study fully changes in healing produced by any influencing factor on the wound it is, therefore, important to know the behaviour of the wound throughout its healing course and not at individual points in the process.

The present investigation was to study the effect of pituitary somatrophic hormone on healing. This substance was isolated in 1945 by Li, Evans, and Simpson. The preparation used in these experiments was made for us by their method by Messrs. Paynes & Byrne. This hormone was chosen for study for several reasons:—

a. It is well known that age has an effect on the rate of healing. Growth hormone would, therefore, appear to be a possible factor which might influence healing.

b. The work of Selye (1951) has shown that somatrophic hormone was antagonistic to the action of cortisone and ACTH on granulation tissue and infections.

c. Prudden, Nishihara, and Ocampa (1958) have shown that growth hormone has a definite anabolic effect after wounding.

EXPERIMENTAL DATA

White male rats weighing 250 g. were used throughout. A control rat and one for each group of experiments were taken from each litter. A standard proprietary rat diet and water were allowed *ad lib*.

The wounds were made under general anæsthesia (avertin) after shaving the hair from the skin. The wounds were 5 cm. in length in the midline of the ventral abdominal wall and were made right through to the peritoneal cavity. Closure was effected by continuous fine nylon sutures in two layers (the sutures were removed before measuring the strength of the wound; monofilament nylon allows this to be done easily without disturbing the wound).

The hormone was given in two ways. In one group of animals a daily injection of 0·25 mg. of pure hormone (equivalent to 2·5 rat units) was given subcutaneously at the time of operation and daily throughout the experiment. In a second group 1·25 mg. of pure hormone was introduced directly into the wound at operation. In both methods the hormone was dissolved in isotonic saline and administered by micro-syringe.

No dressings were used after operation. Animals were all kept in separate cages.

On the appropriate day the animals were killed with chloroform and the tensile strength was measured using a tensiometer of the pattern described by Sandblom, Petersen, and Muren (1953).

In addition to measuring the tensile strength a portion of each wound was taken for histological examination. Sections were examined by hæmatoxylin-eosin, Mallory and Van Gieson stains.

The thymus was weighed after the death of all the animals. It was found that those receiving the hormone by either route had two to three times as much thymic tissue as the controls, thus proving the potency of the preparation used (Geschwind and Li, 1955).

RESULTS

The results of the three series are shown in the accompanying graphs (*Fig.* 102). It is obvious that no shortening of the latent interval occurs in the animals treated with the hormone, nor is any increase in the rate of development of tensile strength to be seen of sufficient degree to have clinical application.

Histological examination of the wounds failed to show any difference between the two treated series and the controls. In all animals macroscopical appearances are the same at any individual point by time in the healing process.

From these observations it would appear that neither clinical change in the form of collagen deposition nor cellular change is greatly influenced by the administration of somatrophic hormone, even when it is given in large local dosage.

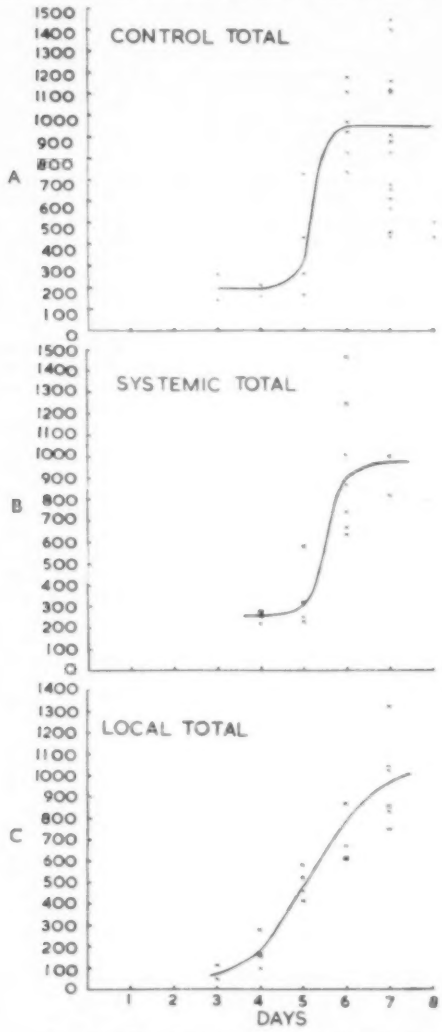

FIG. 102.—Each × represents one animal. The strength is measured in terms of the tension needed to disrupt fully 1 cm. of a standard abdominal wound in all its layers (i.e., total bursting strength).

It is, therefore, unlikely that administration of this hormone will have a practical surgical value except in cases where a deficiency of pituitary function exists in growing children.

It has been suggested that preparations of bovine pituitary gland have no effect in human beings. Our preparation was used on 2 patients who received 30–50 mg. daily over a period of two weeks. Urinary steroid studies showed that there was an effect on corticoid metabolism in one of these patients but not in the other. The effect must, therefore, be regarded as a definite but variable one when applied to man; possibly it is dependent on the patient's endocrine balance at the time of administration.

The reason for speedier healing in young animals still remains unexplained. We have recently inclined to the view that it may be, in some measure at least, an index of the difference in the collagen of young animals. It is well known (Orekhovitch, 1952) that there is a much larger proportion of soluble (and therefore more readily mobilized) collagen (or procollagen) in the young animal. We are at present carrying out further experiments based on this premise.

SUMMARY

1. Pituitary growth hormone was administered systemically and locally to male rats on whom laparotomy was performed.

2. No marked change in the latent interval of healing was produced by administration of the hormone.

3. No histological change was detected in the wounds of treated animals.

4. It is unlikely that the hormone will be of practical value except where endocrine abnormality exists.

Acknowledgements.—I should like to thank Mr. d'Abreu and Professor Stammers, who made it possible for me to carry out these investigations and who encouraged and helped me during the work. I should also like to thank Messrs. Paynes & Byrne, who took considerable trouble in preparing the hormone for these experiments.

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COLD-POTENTIATED RECOVERY OF BLOOD-PRESSURE AFTER BLEEDING IN RATS

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WHILE engaged in studying the tolerance of hypothermic rats to bleeding under anæsthesia, it was noted that the blood-pressure response to moderate arterial hæmorrhage differed consistently from that in normothermic rats. Whereas in the normothermic animal after the fall in pressure induced by such bleeding there was little tendency for the blood-pressure to recover, in the cooled animal a brisk recovery of blood-pressure of some magnitude was the rule. It seemed possible that a homeostatic response was being potentiated by cooling, a quite unexpected event, since the general consequence of lowering body temperature is slowing of metabolic tempo. In this paper are presented observations on the potentiation by body cooling of the recovery of blood-pressure following the hypotension of bleeding.

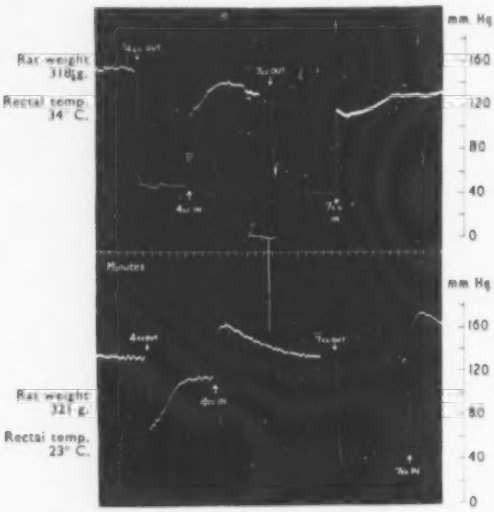

FIG. 103.—Comparison of the effects of single bleeds in warm and cooled rats. The second illustration shows a large recovery after loss of 4 ml. of blood followed by pronounced overswing on replacement. A small recovery follows loss of 7 ml.

METHOD

Rats of several strains and either sex, weighing from 250 to 450 g., were anæsthetized with intraperitoneal pentobarbitone, 7·5 mg. per 100 g. bodyweight, supplemented when necessary by ether inhalation.

Animals were cooled by surface application of crushed ice; body temperature, measured by a rectal mercury thermometer, was lowered to about 25° C., and the ice was then removed. The rectal temperature thereafter remained remarkably steady, gentle warmth being occasionally required to prevent undue further fall. Respiration was spontaneously maintained, but constant watch for tracheal obstruction by mucus was necessary. The rectal temperature also tended to fall in the uncooled anæsthetized control animals, and warmth was often necessary to maintain it at about 35° C.; this level is here referred to as warm or 'normothermic'.

Through a midline neck incision tracheostomy was performed and the left carotid artery cannulated. The carotid sinus regions and vagus nerves on both sides were also exposed by further dissection when necessary. The carotid blood-pressure was recorded directly on a kymograph, the carotid cannula being connected to a recording mercury manometer through a three-way tap. To permit graduated arterial bleeding an inverted 10-ml. glass syringe was attached to the third limb of the tap; 1000 I.U. of heparin solution were injected into the carotid directly after cannulation.

Ordinarily the three-way tap was set to connect the cannula to the manometer and exclude the syringe, so that the blood-pressure was recorded continuously. When the rat was to be bled the tap was turned to connect the carotid cannula to the syringe and exclude the manometer; a pre-determined quantity of blood was then allowed to displace the piston by the force of the arterial blood-pressure in about 30 seconds. Re-setting the tap terminated the blood withdrawal and reconnected the manometer, which at once recorded a fall in pressure. The manometric system was filled with normal saline solution; a fall in pressure of 50 mm. Hg resulted in approximately 0·9 ml. of saline entering the carotid cannula, and conversely, blood entered the connecting tubing when the pressure rose.

The standard method of moderate bleeding adopted was the following: 4 or 5 ml. of blood were withdrawn and a period of 6 to 8 minutes allowed after the fall in blood-pressure so induced for observation of recovery; the blood was then re-injected and the pressure allowed to stabilize again. This sequence of bleeding and re-injection was capable of repetition without significant alteration in the blood-pressure pattern; in 2 test animals 9 such sequences were well tolerated.

Vagotomy was carried out by dividing the previously isolated nerves. Cleaning the carotid bifurcation by dissection was found unreliable in the vagotomized animal as a means of carotid sinus denervation as determined by observing the effect of occlusion of the carotids on the blood-pressure; a more certain effect was obtained by the instillation of 0·5 ml. of 5 per cent procaine into each side of the neck after fully displaying the carotid bifurcations. The vagi, running close by, were presumably also paralysed; hence vagal activity could only be modified alone by dividing the vagi before carotid sinus activity was interrupted. Injection of 5 ml. of local analgesic into another region, the femoral triangle, as a control was without effect on the blood-pressure.

THE BLOOD-PRESSURE RESPONSE TO BLEEDING

The characteristic difference in behaviour between normothermic and hypothermic states is illustrated in the blood-pressure records of two rats of similar weights shown in *Fig.* 103. Each rat was subjected to the standard bleeding procedure, 4 ml. of blood being withdrawn and re-injected 6 minutes later. In the animal at a rectal temperature of 23° C. there was a considerable fall in blood-pressure after bleeding followed by a prompt rise, and re-injection of the blood produced a considerable overswing. In the animal at 34° C. the fall in blood-pressure was greater, and neither spontaneous recovery nor overswing after re-injection took place.

Each animal was then bled 7 ml. In the cooled animal this much larger blood-loss caused the blood-pressure to fall further than before and then show a smaller recovery which was not well sustained; an overswing followed re-injection. In the warm animal, the extent of the blood-pressure fall, the absence of recovery and of overswing were all very like that following the smaller bleed.

The change in response to moderate bleeding brought about by cooling was first noted in rats being bled progressively by separate small amounts of 1 ml. at 2-minute intervals. Under these conditions in the hypothermic animal, the fall in pressure occasioned by each withdrawal of blood tended to be compensated during the interval before the next bleed. After successive withdrawals the vigour of compensation increased for a time and then faded away as the blood-volume became grossly depleted. In the warm animals, on the other hand, there was little compensation between bleeds at any stage; in consequence the rate of fall of blood-pressure was greater. In the later stages of such progressive intermittent bleeding the blood-pressures, then at low levels, differed little in the two states. Examples are shown in *Fig.* 104, the forms of the tracings showing striking differences.

Table I compares the blood-pressures recorded after withdrawal of 5 ml. and 8 ml. of blood by the above progressive intermittent technique in 5 warm and 5 cooled animals. The mean initial blood-pressure in the warm group was 148 ± 7·6 mm. Hg; cooling to about 23° C. lowered the pressure in the cooled group to a mean of 108·8 ± 7·6 mm. Hg. After the loss of 5 ml. of blood, however, the situation was reversed, the cooled animals maintaining a notably higher pressure (mean 73·2 ± 5·7 mm. Hg) than the warm group (mean 41·6 ± 5·7 mm. Hg). By the time 8 ml. had been lost the disparity had disappeared, both

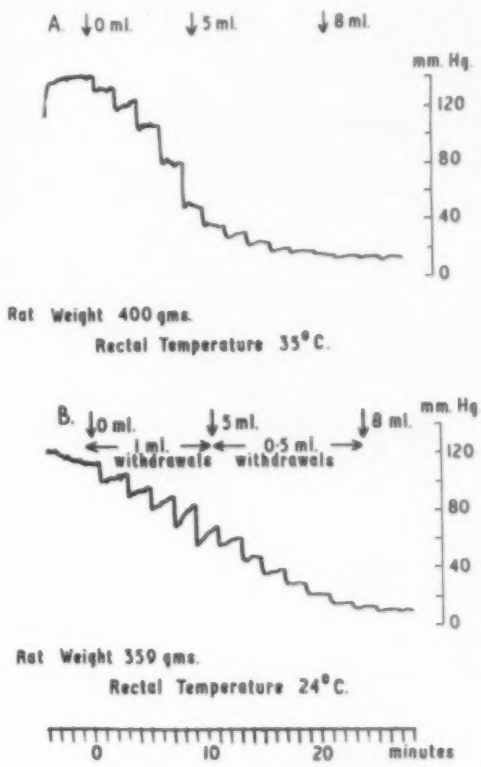

FIG. 104.—Comparison of the effects of repeated small bleeds in warm and cooled rats. The second illustration shows considerable recovery after each bleed until 5·5 ml. have been withdrawn. No tendency to recover is shown by the warm animal.

Table I.—RESPONSE TO PROGRESSIVE INTERMITTENT BLEEDING

	WEIGHT IN g.	RECTAL TEMPERATURE °C.	BLOOD-PRESSURE IN mm. Hg		
			Initial	After 5-ml. Bleed	After 8-ml. Bleed
Normothermic	366	36	120	36	24
	400	34–36	142	50	20
	371	36	154	54	18
	367	37–35	164	28	24
	410	37–34	160	40	46
Mean and standard deviation	383	35·7	148·0 ± 7·63	41·6 ± 5·7	26·4 ± 4·90
Hypothermic	348	24–23	100	88	36
	400	24–23	122	68	24
	359	24–23	110	58	10
	385	24	86	64	30
	345	25–23	126	90	35
Mean and standard deviation	367	23·6	108·8 ± 7·63	73·6 ± 5·7	27·0 ± 4·90

groups having approximately the same mean pressures.

The response to single withdrawals of 5 ml. of blood was next examined; the height to which the blood-pressure recovered above the post-exsanguination level was measured in 6 warm and 9 cooled animals. The results are shown in *Table II*. The mean recovery in the warm group was $2 \pm 3{\cdot}7$ mm. Hg, in the cooled group $36 \pm 3{\cdot}0$ mm. Hg. Further, the cooled group again exhibited the higher carotid pressure at the end of the recovery phase after bleeding, $93 \pm 3{\cdot}9$ compared with $49 \pm 4{\cdot}8$ mm. Hg.

20–60 mm. Hg were obtained. A pressor response also followed temporary respiratory arrest in another such animal.

The effect on the response to bleeding of blocking sympathetic ganglia with hexamethonium bromide and of interrupting the vagi and carotid sinus nerves was next investigated.

MODIFICATION OF THE RESPONSE BY HEXAMETHONIUM BROMIDE

Hexamethonium bromide was administered to 15 rats either by the cannulated carotid artery, the jugular vein, or by intraperitoneal injection. The

Table II.—RESPONSE TO SINGLE 5-ML. BLEED

	Weight in g.	Rectal Temperature °C.	Blood-pressure in mm. Hg			
			Initial	After Bleed (A)	After Recovery (B)	Recovery (B–A)
Normothermic	344	35	166	44	54	10
	373	36	140	30	42	12
	347	36	172	64	56	−8
	360	36	150	38	42	4
	311	35	122	36	30	−6
	348	35	152	72	72	0
Mean and standard deviation	347	35·5	150 ±6·89	47 ±5·48	49 ±4·82	2 ±3·71
Hypothermic	343	23	146	74	100	26
	355	26	134	52	102	50
	341	24	128	44	72	28
	316	25	136	56	100	44
	358	25	138	62	98	36
	382	25	110	48	88	40
	327	24	130	74	98	24
	360	24	112	50	96	46
	345	25	96	58	86	28
Mean and standard deviation	347	24·6	126 ±5·63	58 ±4·47	93 ±3·92	36 ±3·03

Table III.—INTERFERENCE WITH RECOVERY BY GANGLIONIC BLOCKADE

	Weight in g.	Rectal Temperature °C.	Recovery at Third Minute mm. Hg		Interval between Second Bleed and C_6 min.
			Before C_6	After C_6	
	350	24	20	6	7
	340	20	20	0	5
	—	22	34	16	5
	325	22	42	14	7
	350	26	46	36	7
Mean and standard deviation	341·3	22·8	32·4 ±5·42	14·4 ±6·12	6·2 ±0·45

The trifling immediate recovery in the warm animals suggested the possibility of delayed recovery; no such response was encountered in these animals, some of which were observed for periods up to 30 minutes after bleeding. From time to time, however, in other normothermic rats immediate recovery of appreciable magnitude was noted. Determinations of the upper limit of body temperature compatible with cold-potentiated recovery showed a wide range, good recoveries first appearing at very different temperatures in several animals cooled slowly; extremes of 32° C. and 25° C. were encountered. That pressor responses were possible in normothermic animals exhibiting no spontaneous recovery after moderate bleeding was shown by injecting noradrenaline intravenously (1–5 γ); temporary rises of pressure of

rats varied in weight from 311 to 439 g., mean 357 g. In both warm and cooled animals intravascular doses of 2–4 mg. were clearly effective in lowering blood-pressure; doses of the order of 20 mg. arrested respiration. Intraperitoneal injection of 4 mg. in warm animals resulted in large falls in blood-pressure beginning within 1 minute of injection; in the cooled animals, however, there was little change in the prevailing blood-pressure. It was frequently noted that the blood-pressure was unchanged by a second dose of hexamethonium bromide after recovery from an effective first dose; 2–4 mg. by the intravascular or 4 mg. by the intraperitoneal route was therefore taken to be an effective dose for sympathetic ganglionic blockade. Second injections were considered unreliable.

To examine the effect of ganglionic blockade on the standard blood-pressure pattern after bleeding, 5 animals were cooled, the patterns recorded, and the blood re-injected; 4 mg. of hexamethonium bromide were then injected intraperitoneally and 5–7 minutes later the animals were again bled as before. *Fig.* 105 is a typical record showing inhibition of recovery by hexamethonium. *Table III* compares the recoveries obtained in mm. Hg before and after ganglionic blockade; recoveries were measured at the third minute after commencement, when approaching the maximum. The average recovery in these cooled animals was reduced from $32{\cdot}4 \pm 5{\cdot}4$ mm. Hg to $14{\cdot}4 \pm 6{\cdot}1$ mm. Hg by sympathetic ganglionic blockade.

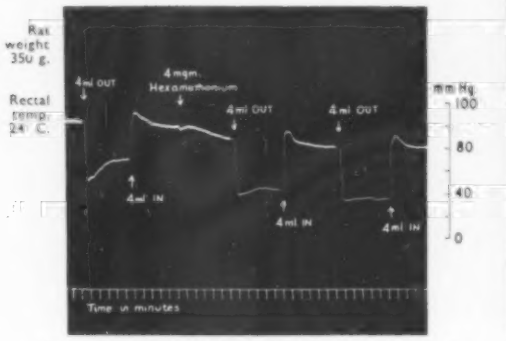

FIG. 105.—Effect of hexamethonium on responses to hæmorrhage in cooled rats. The cold-potentiated recovery is abolished.

It was not to be expected that hexamethonium would much alter the recovery phase in the warm rat; this proved to be the case in 2 animals at 33° C. The systemic blood-pressure fell considerably after injection of the hexamethonium into the peritoneum and in consequence only 3 ml. of blood were withdrawn. No recovery ensued.

Hexamethonium bromide was given to a further 4 cooled and 3 warm animals intravascularly several minutes after bleeding, i.e., after the recovery period. The blood-pressure fell after the injection in all these animals, but the fall was considerably greater in the cooled animals with large recoveries than in the normothermic with but little recovery.

MODIFICATION OF THE RESPONSE BY INTERRUPTION OF VAGI AND CAROTID SINUS NERVES

The procedure was similar to that employed for ganglionic blockade, nerve interruption taking the place of hexamethonium administration in 10 hypothermic animals (*Fig.* 106).

Vagotomy was performed in 8; inconstant restriction of recovery after standard bleeding resulted. In 7 animals the carotid sinuses were denervated using procaine; 5 of these animals had previously been vagotomized and in the remaining 2 the procaine is presumed to have had access to the carotid sinuses and intact vagus nerves simultaneously. Recovery was much reduced. In one of the latter, however, very little change in response followed application of procaine which may therefore have failed to affect conduction; this animal is nevertheless included in the table (*Table IV*).

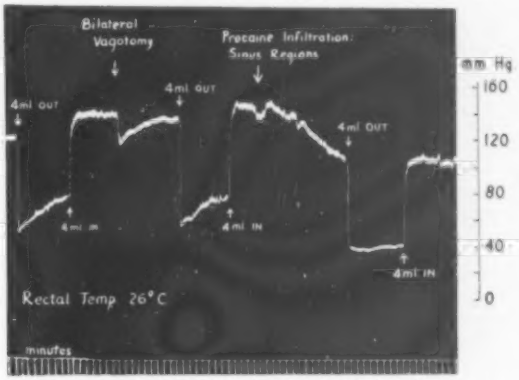

FIG. 106.—Effect of bilateral vagotomy and 'sinus denervation' on responses to hæmorrhage in cooled rats. The cold-potentiated recovery is abolished.

Table IV.—INTERFERENCE WITH RECOVERY BY AFFERENT DENERVATION

	Weight in g.	Rectal Temperature C.	Recovery at Third Minute mm. Hg		
			Before Denervation	After Vagotomy	After Procaine
	300	23	28	—	26
	445	22	12	—	0
	380	25	36	26	4
	350	24	22	18	2
	360	26	24	18	10
	370	24	12	12	6
	395	25	12	2	−6
	270	22	18	6	—
	350	23	6	2	—
	330	23	22	0	—
Mean and standard deviation	355	23·7	19·2 ±2·85	10·5 ±3·36	6·0 ±3·83

DISCUSSION

We have been unable to find reports of the behaviour of the systemic blood-pressure in normothermic or hypothermic rats after bleeding by techniques like our own. The failure of most of our normothermic animals to show any attempt to restore the blood-pressure after the fall of moderate

hæmorrhage was surprising, for the consensus of view in the general literature sees vigorous attempts at restoration of blood-pressure in the earlier stages of bleeding. For example, McDowall (1956) states that even after large withdrawals of blood the pressure usually recovers, and that in the dog the blood-pressure returns to normal within 10 minutes after withdrawal of 2 per cent of the body-weight.

Whatever may be the significance of the failure of recovery in the normothermic animals, there is no question that lowering body temperature permitted very considerable restoration of pressure in the hypothermic rats. *Tables I* and *II* are evidence of this. This cold-potentiated recovery is interfered with by sympathetic ganglionic blockade as shown by *Table III*, which suggests that the recovery is at least in part the result of sympathetic activity. Further evidence of increased sympathetic activity in the cooled animals after bleeding is provided by the observation that the blood-pressure fall is greater in the hypothermic animals than in the normothermic when hexamethonium bromide is given intravascularly at about the end of the recovery period after bleeding. Montgomery, Prevedel, and Swan (1954) have suggested that there is continuous sympathetic overactivity due to carotid sinus activity in the hypothermic dog. In our hypothermic animals carotid sinus activity appeared to be very important in the recovery phase after bleeding, for in 4 out of 5 vagotomized animals application of procaine to the carotid sinuses largely abolished recovery. The denervation, however, did not influence the general blood-pressure level, which is not in keeping with the idea of continuous carotid sinus activity in hypothermia. In some of our animals the vagi also played an important part in establishing the recovery pattern, their division notably impairing it.

These findings suggest that the carotid sinus reflex is largely responsible for the recovery pattern of the cooled animal. Korzuini, Malcolm, and Brooks (1954) have reported augmentation of reflex action directed from somatic nerves in spinal cats when the spinal cords are cooled, the animals themselves remaining normothermic. It is possible that our animals exhibit the same phenomenon of augmentation of reflex activity by cooling, in this instance a visceral reflex or reflexes being concerned.

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SHORT NOTES OF RARE OR OBSCURE CASES

CARCINOMA OF THE PAROTID DUCT

BY D. C. VORA, WREXHAM

CARCINOMA of the parotid duct is a rare condition. Goforth (1927) was the first to report a case. Figi and Rowland (1944) reported 1 case and so did Lyall and Golomb (1954). Beyer, Blair, and Hamilton (1956) reported 2 cases. All put together, 5 cases have been reported so far and all of them in American literature.

The following case would therefore appear to be worth recording:—

Mrs. M. J. D., a housewife, 69 years of age, came to the Out-patient Department of the War Memorial Hospital, Wrexham, on Feb. 6, 1957, with a history of intermittent swelling of the left parotid over the last ten years. About a month previously she had noticed a swelling over the left parotid duct.

On examination a firm swelling was felt in the line of the left parotid duct at the anterior border of the masseter. A provisional diagnosis of parotid calculus was made and the patient sent for X-ray examination.

Radiography did not show any calculus. On sialography the lipiodol entered the papilla of the parotid duct, but did not travel any appreciable distance down the duct.

The patient was seen again in February, March, and June. At the last examination the lump over the parotid duct was very small and it was concluded that the swelling was of an inflammatory nature. The possibility of a neoplasm of the parotid duct did not cross our minds.

The patient returned in November, 1957, with a fleshy swelling at the site of the duct orifice which was firm to touch and looked like a new growth. When, prior to biopsy, the growth was held with a forceps, it was found to have a pedicle coming from inside the duct. The wall of terminal 5 mm. of duct was free, as shown by a probe moved along the wall of that part of the duct. At this stage our interest was aroused because none of us had seen a case of carcinoma of the parotid duct.

Histology showed a squamous-cell carcinoma (*Fig.* 107), but in many places basal-cell proliferation was marked (*Fig.* 108). A diagnosis of squamous-cell carcinoma of the parotid duct was made.

She was referred to Dr. Cole of Manchester for radiotherapy because the histological nature was taken to indicate radiosensitivity. She was treated by radium needles. There has been no recurrence to date.

DISCUSSION

All over the body carcinomata of tubes conducting the secretions or excretions of organs are much rarer than those of organs themselves. This is true for ureters, bile-ducts, and salivary ducts. One probable reason would be the large epithelial surface of glands as compared to tubes. Primary carcinoma of the parotid duct is very rare, and as far as I can trace only 5 cases have been reported so far. Probably the

condition must be commoner than the paucity of case reports suggests, because in many advanced cases site of origin would be almost impossible to decide.

Figi and Rowland (1944) maintain that symptoms of true neoplasm of the parotid and submaxillary ducts are not uniform. As far as carcinomata of parotid duct alone are concerned I have found that they occur in age group 40–70 years and complain

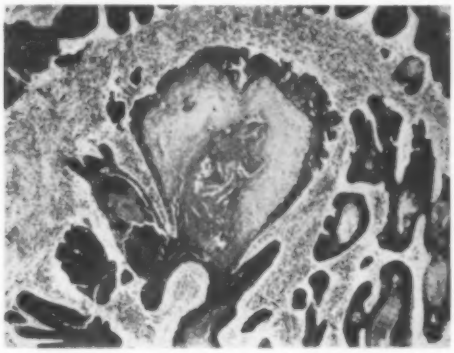

FIG. 107.—Showing proliferation of cells originating from the parotid duct. Groups of squamous cells with keratinization present. P.A.S. (× 27.)

FIG. 108.—Showing groups of cells with basal-cell characteristics. P.A.S. (× 73.)

either of recurrent swelling of the parotid gland or of a lump along the parotid duct, and some complain of both. Goforth's (1927) case did not have a typical history. She presented with pain in the left side of the face but had a "small lump" inside the left cheek for years and it started growing bigger a few months after the onset of pain. The duration of symptoms has been "months" except in one of the cases of Beyer, Blair, and Hamilton (1956), where it was days.

The present case gave a history of recurrent parotid swelling for ten years and of swelling in the line of the parotid duct for one month. If we take the onset of disease to coincide with the appearance of swelling over the parotid duct, a period of eleven months elapsed between onset and diagnosis and a period of one month between onset and first consultation. Probably carcinoma developed in the duct recently and did not have any causative relationship with parotitis.

A nodule in the line of the parotid duct with or without recurrent swelling of the parotid gland should make one look for calculus, and if no calculus is found a biopsy should be performed. The presence of tenderness does not exclude malignancy and even decrease in size under observation does not negative malignancy, as was shown by our case. Neoplastic and inflammatory elements may be associated, as they are in cancer of the colon. The regional lymph-nodes should be examined.

Goforth's (1927) case was a squamous-cell carcinoma with metastases and Lyall and Golomb's (1944) was a squamous-cell carcinoma. Three others were adenocarcinoma. The present case was a squamous-cell carcinoma with a marked basal-cell element. Including the present case there have been equal numbers of squamous-cell and adenocarcinomata. The lining of the parotid duct is of cubical epithelium and hence squamous-cell carcinoma arises by metaplasia.

It is very difficult to hold any dogmatic views on treatment on the basis of so few cases. One of Beyer, Blair, and Hamilton's (1956) cases developed secondaries in the left submaxillary lymph-node five months after operation and then lived for twenty years after suprahyoid block dissection. The primary had been treated previously by surgery and radiotherapy. This case stresses the benefit of an aggressive attack. The present case was treated by radiation alone and to date (eleven months) there has been no recurrence. Most of the clinicians have treated their cases by surgery and radiotherapy.

CONCLUSIONS

1. Primary carcinoma of the parotid duct is a rare condition. One case has been added to 5 reported so far.

2. The symptoms are those of a nodule along the parotid duct and/or repeated parotid swelling. X-ray examination excludes calculus but only biopsy excluded benign lesion.

3. Histologically they are adenocarcinomata or squamous-cell carcinomata.

4. They are slow-growing tumours and by general opinion are best treated by surgery combined with radiotherapy.

My thanks are due to Mr. J. E. Spalding and Dr. M. P. Cole for kind permission to publish the case; and to Dr. L. Wise for histological opinion and photomicrographs.

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MIGRATORY GALL-STONE PRESENTING AS A SINUS THROUGH THE CHEST WALL

By A. J. WEBSTER

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THE following case seems worth reporting because of its rarity. A search of the literature has failed to find an exactly comparable example.

CASE REPORT

HISTORY.—The patient, Mr. T. M., aged 70 years, a retired business man, was first seen in March, 1958. He was complaining of a painful swelling over the right lower ribs about the mid-axillary line, which had been present for six weeks. For four days prior to his being seen pus had been discharging through a small opening.

Previous History.—For some years he had noticed a 'tightness' over the right lower ribs, but this had not worried him particularly. In October, 1957, there was some increased discomfort and he had vomited twice, but the discomfort lasted only a day or two and then settled. He did not complain of indigestion or of flatulence, except in the lower bowel. He had been feeling tired recently and had lost some weight during the last few months. An electrocardiogram four years ago, taken because of precordial pain, had revealed no abnormality.

FIG. 109.—Photograph showing: A, The portion of rib removed; B, Tissue with the sinus track through it; C, The mouth of the sinus with surrounding skin; D, The gall-stone.

On examination in March the patient was a spare but healthy looking man of 70 years. In the mid-axillary line over the eighth, ninth, and tenth ribs there was a swelling discharging pus from a central sinus and in appearance like a pyogenic abscess. There were a few palpable glands in the right axilla. A radiograph of the region revealed no suggestion of underlying bone infection.

He was admitted to hospital on March 17, 1958, and on the following day, under general anæsthesia, what proved to be a large abscess was incised and a quantity of pus evacuated. The tissues overlying the abscess were very œdematous but the abscess appeared to be superficial and no connexion with deeper structures could be found. The wound, which was left open, discharged pus freely at first, but by March 23 was apparently healed. A day or two later, however, it broke down and discharged more pus.

Bacteriological examination of the pus revealed no organisms in direct films and no growth on culture. Further culture for tuberculosis was reported negative on May 27.

He was discharged to the care of his own doctor on April 6 and was seen at intervals thereafter until May. A small sinus persisted with a slight purulent discharge.

FURTHER COURSE.—He ceased to report after May and was not seen again until September, at which time the sinus, which had never healed, was still present and there was still some thin pus emerging. He was readmitted on Sept. 25, 1958, and a radiograph showed a focus suggesting a low-grade osteomyelitis of the eighth rib in the anterior axillary line. On Sept. 30 the area was explored again through an elliptical incision surrounding the sinus, the track of which appeared to lead down to the upper margin of a rib and beyond. The rib adjacent to the track looked unhealthy and a portion of it was excised. At this stage some brown amorphous material was noticed in the wound. On further exploration it was discovered that this was coming from a large gall-stone 6 cm. long by 3 cm. in diameter, roughly cylindrical in shape, lying deep to the rib, with one end pressing against it. The appearance of the stone suggested that it was mainly composed of pigment. It was lying in an abscess cavity which, as far as could be discovered, had no orifice other than the sinus. The stone was removed, but it was thought wise to do nothing more than to insert a drainage tube and to close the wound around it.

The drainage tube was removed in 48 hours and thereafter there was some thin purulent discharge from which coliform bacilli, sensitive to chloramphenicol, were grown. Chloramphenicol was accordingly administered and the wound rapidly dried and healed. He was discharged on Oct. 26.

A cholecystogram on Oct. 14 showed a non-functioning gall-bladder 12 and 13 hours after biliodyl. No opaque gall-stones were seen.

The pathologist reported that the sinus track was lined with highly vascular granulation tissue. Bile-pigment was present in the walls and in macrophages. Frozen sections revealed large numbers of crystals of fatty acids and needle-shaped cholesterol crystals.

DISCUSSION

Cases of gall-stones rupturing from the gall-bladder into the bowel are not uncommon and cases of gall-stones found in the general peritoneal cavity, the stomach, and even in a renal pelvis, have been reported. Grimes and Steinbach (1955), from San Francisco, have described a case where a sinus in the anterior abdominal wall, appearing after trauma, led to a gall-stone encysted in the abdominal wall and then directly into the gall-bladder.

In the present case the absence of previous history seems remarkable. The stone would appear to have

ruptured from the gall-bladder and to have tracked to the position in which it was found without causing noticeable symptoms, unless the incident of increased discomfort and vomiting was coincident with the rupture. Presumably this absence of signs and symptoms might have continued had not infection supervened, leading to abscess and sinus formation. No communication between the abscess cavity and the gall-bladder was found and no bile leaked out after the second operation, suggesting that any communication between this cavity and the gall-bladder had become closed.

SUMMARY

The case of a patient presenting with a chronic sinus in the chest wall which was found to connect with an abscess cavity containing a gall-stone lying deep to the eighth rib is described. After excision of a portion of rib and the removal of the gall-stone, recovery seemed to be complete.

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A CASE OF LOCALIZED GASTRIC ABSCESS

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GASTRIC abscess is frequently described in the literature under the heading of 'Phlegmonous gastritis' (Lyall, 1938; Eliason and Wright, 1938; Vass and Sirca, 1941). It is clear, however, that under this general term there are at least three pathological entities—namely, diffuse suppurative gastritis, diffuse gastric cellulitis, and localized gastric abscess. Notwithstanding these morbid anatomical differences, the basic underlying causes are probably the same or closely related.

The rarity of gastric abscess is remarkable, bearing in mind the frequency of peptic ulceration, gastric neoplasia, and the dietetic insults with which civilized man confronts his stomach. (Novak, 1919, stated that the first recorded description is attributable to Galen, while the earliest relatively complete accounts belong to the eighteenth century (Verandaeus, Borel, Sand, and Boret).) At the time of writing his paper he considers that not more than 25 cases of circumscribed gastric abscess have been recorded. Since then, however, relatively few cases have been reported, and Kellert (1936) stated that the sum total of recorded cases of phlegmonous gastritis was about 150. It is unlikely that these figures are accurate, since only two years later Eliason and Wright (1938) found in the literature a total of 307 cases of phlegmonous gastritis, including 31 of their own. Out of these, 7 were localized lesions. Parsonnet, Fishler, and Sanger (1951) could find reports of only 3 localized abscesses between 1931 and 1951, so that, although there seems to be discrepancy between one author and another, this lesion is quite clearly an uncommon pathological entity. The further example presented as a very common clinical syndrome.

CASE REPORT

HISTORY.—Mr. L. C., aged 48 years. This patient was admitted as an emergency on Dec. 10, 1956, with a 24-hour history of upper abdominal pain associated with repetitive vomiting. The pain fluctuated in intensity but had been present most of the time and did not suggest a true colic. The vomit was unremarkable, but contained traces only of bile. The patient denied any previous dyspeptic upset, although his wife stated that he had occasionally had some slight indigestion after meals for a few months.

ON EXAMINATION.—The temperature was 99·2° and the pulse 80. There was no abnormality to be detected on clinical examination of the chest; the abdominal contour was normal, but there was tenderness and rigidity in the right upper quadrant of the abdomen. Rectal examination was non-contributory.

FIG. 110.—Macroscopica appearance of gastrectomy specimen—opened down the greater curvature. Note the abscess cavity at the left-hand end of the specimen.

The provisional clinical diagnosis was that of acute peptic ulceration or an acute exacerbation of a chronic peptic ulcer which had produced minimal symptoms in the past. The tenderness was thought to be accounted for by peri-ulcerous cellulitis which commonly occurs in these cases, and it was intended to treat him by means of bed rest, sedatives, and possibly an intragastric milk drip.

This schedule was interfered with as a plain film of the abdomen was taken and this was reported on as showing a single distended loop of small intestine which had the appearance of internal strangulation. In view of this, laparotomy was carried out on Dec. 10, the day of admission.

AT OPERATION.—No evidence of intestinal obstruction was found, but a mass the size of a walnut was palpable on the greater curvature of the pyloric antrum. The tumour was accommodated within the substance of the antral wall and there was some œdema of the overlying peritoneum.

Immediate partial gastrectomy was carried out in the belief that this was a benign tumour of the stomach which had presented with symptoms possibly as a result of recurrent intussusception into the pylorus. The post-operative convalescence was uneventful in every way and the patient was discharged on the eleventh post-operative day.

PATHOLOGICAL REPORT.—A partial gastrectomy specimen measuring approximately 16 cm. along the greater curvature and 7 cm. along the lesser. At the extreme distal end of the specimen on the greater curvature is a globular tumour of about 4·0 cm. diameter, which is projecting out from the curvature and into the lumen of the stomach.

On opening, the tumour is a necrotic cystic lesion filled with purulent material and having a wall varying from 1·0 cm. down to 0·2 cm. in thickness. It is juxta-pyloric in situation. The mucosa generally, apart from some œdema in the vicinity of the tumour, appears normal.

Microscopical: This is a pyogenic abscess deep within the muscularis of the stomach wall, associated with small fragments of foreign material which, it is thought, may be of vegetable origin.

DISCUSSION

Gastric abscess (localized phlegmonous gastritis) occurs most commonly about middle life, although Novak's (1919) case was only 19 years old and he quotes Leith as stating that the extremes in age incidence lie between 10 and 85 years. Most authors say that it occurs more commonly in men than women, and it is hardly surprising that in the majority of cases the abscess complicates a pre-existing gastric lesion. The apparent basic causes of the disorder may be classified as follows:—

1. Peptic ulcer (Ogilvie and Scorer, 1929; Eliason and Wright, 1938).
2. Chronic hypertrophic gastritis (Vass and Sirca, 1941).
3. Chronic gastritis due to alcohol or other chemicals (Eliason and Wright, 1938).
4. Penetration by foreign material (Lyall, 1938).
5. Gastric neoplasm: (*a*) Carcinoma (Kellert, 1936); (*b*) Adenomyoma (Petrowitch and Davidowitch, 1933).

It is generally agreed by previous writers that staphylococci and streptococci are the two commonest organisms associated with the abscess, but pneumococci (Parsonnet and others, 1951), *Escherichia coli*, *B. proteus*, *B. subtilis*, and *B. welchii* have all been incriminated from time to time (Novak, 1919; Ogilvie and Scorer, 1929; Rankin and Miller, 1931; Lyall, 1938).

A study of the morbid anatomy and histology of the lesion shows that it usually starts as a suppurative inflammation arising in the relatively loose sub-mucosal connective tissue between the mucosa and the muscularis. The anatomical extent of the lesion varies according to local factors on the one hand, and the general condition of the patient on the other. It is apparent in Eliason and Wright's (1938) collected cases that where there is evidence of generalized systemic infection the gastric lesion is diffuse, or extensive though localized. In the majority of reports the lesion is either close to the pylorus or in the distal half of the stomach (Kellert, 1936; Eliason and Wright, 1938; Lyall, 1938, Parsonnet and others, 1951), and in the majority of fatal cases there is evidence of peritonitis.

The correct clinical diagnosis has almost invariably proved difficult; Rankin and Miller (1931) assert that there is no typical clinical picture; their own case, together with that of Novak (1919) and Lyall (1938), was diagnosed as cholecystitis. Vass and Sirca (1941) diagnosed an abscess as pyloric carcinoma, while Ogilvie (1929) thought that radiologically his case was one of peptic ulcer, although clinically it was carcinoma.

The prognosis in the majority of both diffuse and localized cases of suppurative gastritis has been remarkably bad, death occurring (as a result of peritonitis) from shortly after admission to hospital (Lyall, 1938) up to the twentieth day (Rankin and Miller, 1931). Novak's (1919) localized case, however, made a good recovery following gastrectomy, while Guzzetta and Southwick (1947) successfully treated a diffuse suppurative gastritis by gastrectomy and drainage combined with penicillin and sulphadiazine. Our own patient, having an entirely localized lesion, with minimal peritonitis, made an uneventful recovery following partial gastrectomy performed less than 40 hours after the onset of symptoms. This rapid surgical intervention, undertaken on account of misleading radiological signs, was undoubtedly responsible for the satisfactory conclusion to what might have been a very grave illness.

SUMMARY

1. A case of simple gastric abscess, of foreign-body origin, presented in the manner of an acute peptic ulcer or peri-ulcerous cellulitis.

2. Owing to a radiological appearance suggestive of internal strangulation of the intestine, early surgical intervention led to a partial gastrectomy, before peritonitis had time to complicate the issue. An uneventful recovery was made.

The authors wish to express their thanks to Mr. Cuthbert Roberts, F.R.C.S. (under whose care this patient was admitted), for permission to publish this case.

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JEJUNOJEJUNAL INTUSSUSCEPTION FROM SECONDARY FIBROSARCOMA

By MICHAEL C. OLDFIELD and GEOFFREY WILSON

FROM THE GENERAL INFIRMARY AT LEEDS

THE case here recorded seems worthy of note because of the rather unusual presentation and because it illustrates well the pathological concept of blood-borne metastasis.

CASE REPORT

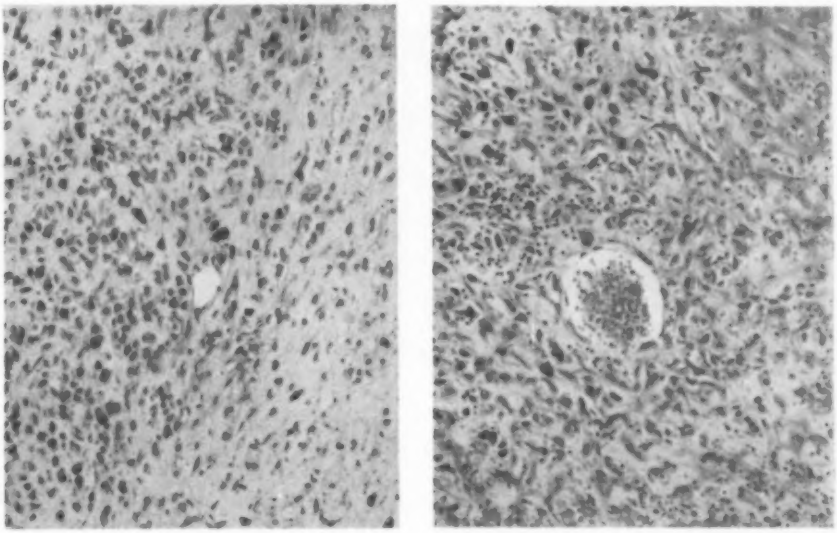
A 55-year-old male patient attended hospital in November, 1950, with a swelling of the left buttock of one month's duration. The swelling was described as "a firm, smooth, freely mobile, subcutaneous tumour, above and medial to the greater trochanter". Surgical treatment was advised but declined by the patient.

He was re-admitted to hospital one year later in November, 1951, because of dull, aching pain in the left buttock, the swelling having increased in size. At operation about half of the gluteus maximus muscle was resected and proved to contain an apparently well-encapsulated tumour. Histological examination showed the tumour to be a fibrosarcoma; irregularity of cells and frequent mitoses indicated a high degree of malignancy (*Fig.* 111 A). There was at that time no evidence of pulmonary metastases. A course of deep X-ray therapy was given to the buttock.

The patient remained well until June, 1956, when he complained of a "sensation of pressure" in the left side of his neck. No clinical abnormality was found, but a radiograph of the chest (*Fig.* 112) showed multiple secondary deposits in the right lung field. Palliative deep X-ray treatment was given to the chest.

Thirteen months later the patient was again re-admitted to hospital with severe upper abdominal colic and vomiting of one week's duration. Physical examination revealed gastric distension, visible peristalsis, and a marked succussion splash. He was treated with gastric suction and intravenous fluids and his symptoms subsided within 48 hours. A barium meal, however, showed abrupt narrowing in the region of the duodenojejunal flexure (*Fig.* 113). The pulmonary metastases were more numerous than a year before.

Exploratory laparotomy was undertaken through a right upper paramedian incision. There was a large sausage-shaped mass in the upper abdomen which proved to be a reducible jejunojejunal intussusception. At the apex, which was just distal to the duodenojejunal flexure, a tumour was palpable within the bowel lumen. This segment was resected and end-to-end anastomosis performed (*Fig.* 114).



A B

FIG. 111.—A, Anaplastic fibrosarcoma of buttock with cellular irregularity and frequent mitoses. (× 128.) B, Secondary fibrosarcoma of jejunum showing similar cellular characteristics to the primary tumour. (× 128.)

Histological examination of the tumour showed it to be a cellular anaplastic fibrosarcoma (*Fig.* 111 B) similar to the tumour removed from the buttock six years before.

The patient made an uninterrupted recovery and was discharged from hospital three weeks later.

DISCUSSION

The frequency of intestinal metastases is difficult to assess since rarely is the whole intestine subjected to careful microscopical examination at necropsy. In a series of 500 cancer necropsies Willis (1952) found intestinal metastatic deposits in 10 cases (2 per cent). He also collected from the literature 135 cases of metastatic growths in the intestines; the primary growths were most frequently melanoma (45 cases), carcinoma of lung (17 cases), carcinoma of breast (12 cases), chorionepithelioma (12 cases),

carcinoma of kidney (10 cases), and carcinoma of stomach (9 cases). The jejunum was most frequently involved.

The most frequent complication of secondary neoplastic disease of the intestines is intestinal obstruction, whether the bowel is invaded from

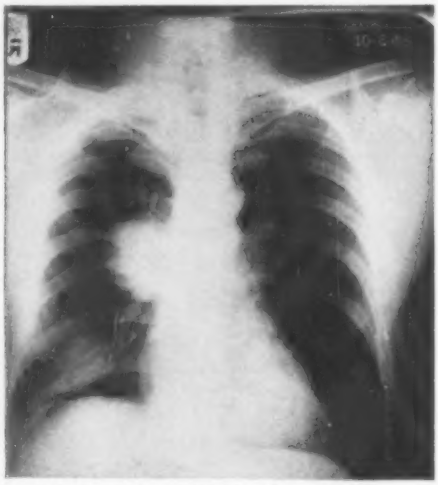


FIG. 112.—Cannon-ball secondary sarcomatous deposits in right lung.

FIG. 113.—Barium meal showing obstruction at the duodenojejunal flexure.

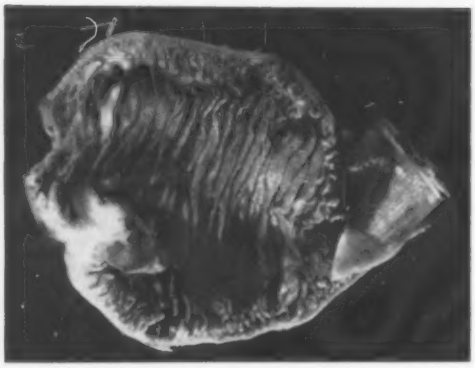


FIG. 114.—The pedunculated secondary fibrosarcoma causing intussusception.

without by tumour, or whether the tumour is truly metastatic. Obstruction due to blood-borne secondary deposits may produce annular stricture, as in the two cases reported by Barnard and Elliot (1930) when the primary tumours were in the lung. More usually, however, the obstruction results from the intussusception of a pedunculated tumour, and occasionally the secondary tumour may be passed in the fæces, as in the case of a hypernephroma recorded by Bevan (1904). Necropsy was not recorded, however, and direct invasion of the bowel cannot be excluded.

Secondary melanoma of the intestine causing intussusception is rare, but instances are recorded by Maxwell (1928), Ross (1930), and Wilbur and Hartman (1931). We found only 1 reference to secondary sarcoma of the intestine causing intestinal obstruction (Waring, 1930). This patient was a girl of 14 years, operated upon for intestinal obstruction and peritonitis, who was found to have a gangrenous mass in the small intestine, which was resected. Two years before this she had had a sarcoma of the upper jaw treated by deep X-ray therapy. Comparison of the intestinal mass and the jaw biopsy showed that both were round-cell sarcomata.

Additional features of interest in our patient were his relatively good health for over a year with known lung secondary deposits and the latent period before the occurrence of the intestinal metastasis.

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TRAUMATIC ARTERIOVENOUS FISTULA INVOLVING THE AORTA

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TRAUMATIC arteriovenous fistula affecting the aorta is an uncommon lesion, and its occurrence in childhood is very rare. We report a case with some unusual features, the management of which posed some problems of interest.

CASE REPORT

The patient, a boy aged 2½ years, was admitted as an emergency three hours after accidental wounding by a ·22 rifle bullet fired at a range of 500 yards. The bullet had traversed the left arm just above the elbow without damaging bone or other major structures, and entered the left chest in the posterior axillary line at the ninth intercostal space. The bullet itself was readily palpable subcutaneously over the ninth rib in the mid-axillary line on the right side. He was in peripheral circulatory failure, blood-pressure 65/45 mm. Hg. There was tenderness and muscular rigidity, most marked in the upper abdomen, and bowel-sounds were absent. Chest radiographs were negative. Resuscitation was accomplished with 150 ml. dextran and 650 ml. blood. Laparotomy revealed that the bullet had entered the abdominal cavity to the right of the second part of the duodenum, the lateral edge of the duodenum showing a small laceration. In this area there was a moderate-sized retroperitoneal hæmatoma. The bullet had then traversed the liver from its inferior to its right lateral surface and there escaped from the abdomen. Other viscera were normal and active bleeding had ceased. There was no evidence of retroperitoneal hæmorrhage elsewhere. The lacerated duodenum was repaired and the abdomen closed with drainage. The bullet was readily removed through a small skin incision over it.

His post-operative circulatory state remained good. He had a paralytic ileus for five days, but thereafter made a steady recovery. Nine days after admission radiography revealed no lesion of the thoracolumbar spine or chest, and movements of the diaphragm were normal.

He returned home on the thirtieth post-operative day. He did not attend for follow-up examination as requested.

The child came under observation again 1½ years after his original admission. His mother complained of his

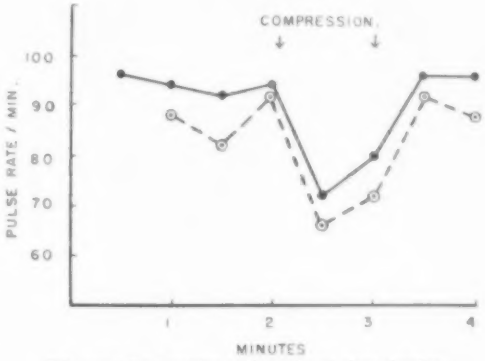

FIG. 115.—Pre-operative radiography of chest showing marked cardiac enlargement.

FIG. 116.—The effect of compression of the fistula on the pulse-rate.

FIG. 117.—Flat plate of abdomen showing area of calcification to left of second lumbar vertebra, which later proved to be the site of a simple traumatic aneurysm.

having attacks of screaming, usually at night, episodes of cyanosis of his face lasting about an hour with no identifiable precipitating cause, intolerance of cold, and breathlessness on bending.

ON EXAMINATION.—He was a well-developed boy of normal behaviour with highly coloured cheeks, which were at times blue, but showing no central cyanosis even on exertion. The pulse-rate was 74 per minute, collapsing in character, and the blood-pressure 125/55 mm. Hg in the arms and 120/50 mm. Hg in the legs. There was no evidence of cardiac enlargement. A loud systolic murmur was best heard down the left sternal border and at the apex. There was no diastolic bruit.

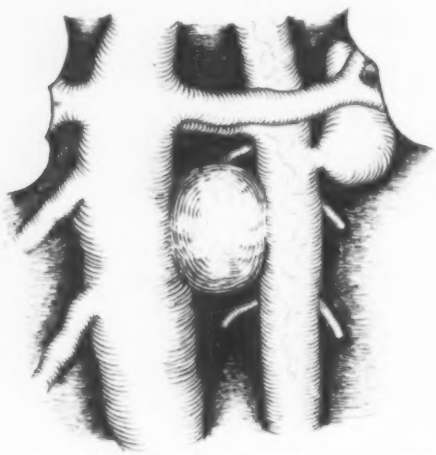

FIG. 118.—Diagrammatic representation of operative findings. The inferior vena cava and lumbar veins are greatly enlarged.

A continuous murmur with systolic accentuation was widely present over the abdomen and back, being loudest in the epigastrium and over the upper lumbar spinous processes, but audible even over the sacrum and iliac crests: indeed the systolic component could be detected in the lower thighs over the line of the femoral vessels. A fine continuous thrill was palpable just to the right of the midline in the upper epigastrium. This was at times difficult to feel, but was maximal three fingerbreadths below the xiphisternum. Deep pressure in this area, without affecting the femoral pulse, resulted in a rise of blood-pressure in the right arm from 125/60 to 130/85, and also in bradycardia (*Fig.* 115).

There was no enlargement of liver or spleen, no ascites, and no visible distension of superficial abdominal veins. There was no evidence of venous or arterial insufficiency in the legs. All the peripheral pulses were palpable.

Table I.—PERCENTAGE OXYGEN SATURATION

Right atrium	79
Upper end of inferior vena cava	79
1 in. above anticipated level of fistula	82·5
1 in. below anticipated level of fistula	76
Lower end of inferior vena cava	60·5
Common iliac vein	62·5

(The 'anticipated level' was the lower border of the 2nd lumbar vertebra.)

Radiological examination of the chest showed generalized cardiac enlargement with displacement of the œsophagus (*Fig.* 116). X-ray examination of the abdomen revealed a circular area of calcification to the left and in front of the body of the second lumbar vertebra (*Fig.* 117) and overlapping the left renal pelvis. On intravenous pyelography there was no deformity of either pelvicalicial system and concentration was good and equal in both kidneys.

A barium swallow and meal failed to disclose any œsophageal varices, and there was no abnormality on proctoscopy. Hæmoblogin estimation was 12·3 g. (86 per cent). Electrocardiographs showed 'splintering' of the R wave in V, suggesting a partial right bundle-branch block.

FIG. 119.—Post-operative radiograph of chest showing considerable reduction in cardiac size.

A diagnosis of a traumatic arteriovenous fistula was made, the abnormal communication being probably situated between the aorta and the inferior vena cava.

The patient was seen on various occasions over the next two years. Further chest radiographs suggested some increase in the degree of cardiac enlargement, but three and a half years after the original injury there were no signs of cardiac failure nor of venous or arterial insufficiency in the legs, nor of dilatation of superficial abdominal veins. He was readmitted for further observation with a view to surgical treatment.

INVESTIGATIONS.—Retrograde catheterization of the aorta and vena cava was carried out: the direct aortic pressure was 133/56 (mean 90) mm. Hg. For technical reasons aortography was unsatisfactory. The right atrial pressure was 12/7 mm. Hg. Continuous pressure recordings, as the catheter was withdrawn, gave only a gradual rise of pressure from the right atrium to the right common iliac vein. Percentage oxygen saturation of samples at several levels are shown in *Table I.*

These results confirmed the presence of a considerable arteriovenous shunt, most probably aorto-caval, but possibly between the left renal vessels. Operation was accordingly advised.

AT OPERATION.—Laparotomy (G. E. M. and W. A. A.) was undertaken with general hypothermia induced by

surface cooling. A bilateral subcostal incision was used, dividing both recti and extending into the lateral abdominal muscles on the left. A calcified mass 4 cm. in diameter was present to the left of the aorta. It exhibited only transmitted pulsation. The maximal area of widespread continuous thrill lay to the right of the aorta. The posterior parietal peritoneum was opened and the duodenum mobilized to the right. The inferior vena cava was greatly enlarged and streams of arterial blood could be seen through its wall, passing both up and down its lumen from an arteriovenous aneurysm which lay between it and the aorta (*Fig.* 118).

The bradycardia noted on compression of the fistula pre-operatively was confirmed on occluding the fistula with the fingers through the intact wall of the vena cava. When this was done with the child's rectal temperature at 32·5° C. nodal rhythm was immediately produced, which rapidly reverted to sinus rhythm on removing the finger. This was repeated several times. Later, however, at a rectal temperature of 34·5° C., digital occlusion of the fistula, while still resulting in cardiac slowing, did not produce any arrhythmia.

The left renal vein was freed and displaced upwards and the aorta and vena cava were mobilized and occluded above and below the aneurysms but below the renal vessels. A pair of lumbar arteries and several large lumbar veins had also to be controlled.

The aneurysm was then opened and dissected free from the great vessels. The calcified mass on the left proved to be a simple aneurysm almost completely filled with partially calcified old thrombus with an opening into the aorta 5 mm. in diameter. The arteriovenous aneurysm was about 3 cm. in diameter, with some calcification in its wall, and communicated with the aorta through an orifice 7 mm. in diameter and with the inferior vena cava through an orifice 15 mm. in diameter (*Fig.* 118). The two defects in the aortic wall were closed longitudinally and that in the inferior vena cava transversely, using interrupted and continuous arterial silk. The traumatic aneurysms were not excised. The aortic and cavai occlusion time was 75 minutes. During the operation the patient received 400 ml. of blood.

His post-operative course was complicated by a mild upper respiratory infection from the twelfth to the seventeenth post-operative day. A chest radiograph showed that by the third post-operative day the heart size was considerably reduced (*Fig.* 119). He was discharged from hospital thirty days after operation.

FOLLOW-UP.—When seen one month later, his general health was much improved. He was normally active, but occasionally tired after prolonged walking. His facial colour was normal. His mother thought he had some swelling of the left leg but this had not been confirmed by the family physician. He had, however, developed visible subcutaneous veins over his lower abdomen, the blood-flow being in an upward direction. There was no peripheral œdema and his exercise tolerance was good. Apart from a faint localized systolic bruit at the site of the previous right femoral arteriotomy, there were no audible murmurs either over the precordium or in the abdomen. The heart size remained considerably reduced. All peripheral pulses were normal. The blood-pressure was 116/76 mm. Hg. Electrocardiographs were normal.

Nine months after operation the patient's condition was satisfactory. He had no complaints. The enlarged veins on his lower abdominal wall were still apparent, but had not increased in size or quantity.

It was evident that he had developed at some time post-operatively a thrombosis of the inferior vena cava. This was probably related to the caval suture line and the site of the fistula, for, as previously stated, the vein wall at this point was grossly thickened and in part calcified. In retrospect it would seem that thrombosis of the inferior vena cava was a likely post-operative complication which might have been prevented by prophylactic anticoagulant therapy.

DISCUSSION

Pre-operatively, exact determination of the location of the vascular anomaly presented some difficulty. The characteristic and widespread murmur left little doubt as to the presence of an arteriovenous fistula, and this was confirmed by the rise in the blood-oxygen saturation in the vena cava about the suspected level. Signs of venous insufficiency are usually noted in arteriovenous fistulæ involving the inferior vena cava (Billig and Shadle, 1957; Branham, 1890; Decker, 1950 a, b). Surprisingly in this case there were no such signs, raising the possibility of the fistula being in the portal system. However, the absence of splenomegaly, ascites, or any evidence of portal hypertension made this unlikely (Decker, 1950 a, b).

The track of the bullet, as estimated by the wounds of entry and exit, together with the calcified hæmatoma, suggested that the fistula lay at or above the renal vessels or perhaps involved them. Venous blood-oxygen levels in the inferior vena cava could not be expected to allow of an exact location, in view of the possibility of retrograde flow of arterial blood, which was indeed subsequently observed at operation. As regards arteriography, it may be extremely difficult to obtain a clear picture in the presence of an arteriovenous shunt, particularly if the latter is large or is associated with an arteriovenous aneurysm. As in this case, it is common to find a simple traumatic aneurysm associated with an arteriovenous fistula (Shumacker, 1948). It may be calcified and thus it can be precisely localized. However, it is well known that such a simple aneurysm cannot be taken as a guide to the site of the fistula (Pemberton, Seefeld, and Baker, 1946), for as in this instance it may prove to be well away from the communication. Failure to localize the fistula below the renal vessels necessitated the use of hypothermia, which could only be discontinued when it became clear that the fistula could be closed without occluding the aorta above the renal vessels.

The appearance of nodal rhythm on occluding the fistula at low body temperature during operation is of interest. It has been shown experimentally that the bradycardia described by Branham (1890) and Nicoladoni (1875) is a reflex phenomenon, both its afferent and efferent components being subserved by the vagus and being abolished by full atropinization of the animal or by denervation of the carotid and aortic pressure receptors (Nicoladoni, 1875). It is possible that hypothermia in this child potentiated the vagal effects of occlusion of the fistula resulting in nodal rhythm.

Comment.—While traumatic abdominal aorto-caval fistulæ have been reported from time to time, only recently has surgical cure been achieved. Freeman and Storck (1947), Pemberton and others (1946), Decker (1950 a, b), and Schafer and Hardin (1952) each reported a single case in which they had successfully closed a traumatic abdominal aortocaval fistula. Their 4 patients were in their third and fourth decades. We can find no report of closure of such a fistula in a child.

It is remarkable that this child survived the original injury. The bullet had entered the aorta

posteriorly on the left and crossed the aortic lumen, emerging through the right side of the aorta and damaging the inferior vena cava before lacerating the duodenum and transversing the liver to lodge in the lower chest wall. The severe hypotension that ensued presumably allowed the arterial hæmatoma to clot and thus arrest active bleeding from the aorta. The relatively small amount of retroperitoneal bleeding found at the initial laparotomy is a witness to the efficacy of this process.

The time at which the fistula became established is a matter for conjecture. It would seem extremely unlikely that it formed in the early hours after injury as no bruit or thrill was detectable at the first operation. No sign of a fistula was elicited in the ensuing week when abdominal auscultation was frequently carried out during the period of paralytic ileus.

The prognosis called for careful consideration. The child showed no signs of cardiac distress other than radiological cardiac enlargement and equivocal electrocardiographic signs and no evidence of arterial or venous insufficiency in the lower limbs. It was felt, however, that the cardiac effects were likely to prove progressive. Furthermore, the possibility of the development of endarteritis or endocarditis (Lillehei, Bobb, and Visicher, 1950) had to be borne in mind. Thus an attempt at radical cure was indicated. In the event the degree of cardiac enlargement was much more than actually suspected. This enlargement subsided within 72 hours of operation and possibly much sooner, indicating that it was probably related to an increase in circulatory blood-volume and cardiac filling. It is unfortunate that no blood-volume estimations were carried out.

In retrospect, it is of interest that the symptoms produced by this large fistula were mild as compared with the degree of cardiac disability and circulatory deficiency reported in adults with comparable lesions. That the child was able to compensate so well is attributable to the ample cardiac reserve and greater circulatory adaptability at this age.

SUMMARY

The closure of a traumatic arteriovenous fistula between the aorta and the inferior vena cava in a child is described.

The rarity of the opportunity to effect such a repair is noted, bearing in mind the chances of survival from the initial injury.

The importance of trying to establish an exact anatomical pre-operative diagnosis is stressed.

It is a pleasure to acknowledge our indebtedness to Mr. N. J. Logie, who referred the child for investigation of the arteriovenous fistula with a view to possible surgery. We also wish to thank Dr. E. Priest for her assistance during investigation.

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LYMPHANGIOMA OF THE MESENTERY OF THE ILEUM

BY J. C. WALKER AND J. A. COOPER

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WHILE tumours of the mesentery are uncommon, lymphatic tumours in this site are extremely rare. The following report describes such a case occurring in the mesentery of the ileum. It was found at operation, but its nature was not clear until histological examination was made.

CASE REPORT

A male, aged 32 years, employed as a trumpet player in an orchestra, was admitted to Dunoon Hospital in the early hours of the morning, complaining of severe colicky abdominal pain and vomiting of some five hours' duration. For some days before the onset of the pain he had experienced discomfort in the region of the umbilicus, excessive abdominal borborygmi, and nausea. This discomfort he attributed to "something he had eaten". The bowels had not moved for 48 hours although flatus had been passed; micturition was normal. Before this attack his general health had invariably been good and he had not previously sought medical advice.

Examination showed the patient to be a well-nourished young male of healthy appearance; his tongue was clean, and pulse, temperature, and respirations were normal. Several attacks of colicky abdominal pain and vomiting occurred during examination. The abdomen was slightly distended, but between the attacks of colic a mobile tumour about 14 cm. in diameter was palpable in the region of the umbilicus. This mass was slightly tender but very mobile, being easily displaced to either right or left lower abdominal quadrant, and in respect of this mobility reminiscent of an ovarian cyst. Rectal examination was negative.

Hæmoglobin was 90 per cent (Tallquist); R.B.Cs., 5·2 million/c.mm.; W.B.Cs., 5400/c.mm.; E.S.R., 4 min. in the first hour.

A straight radiograph of the abdomen did not show fluid levels. No firm diagnosis was made, but it was felt that the obstructive symptoms and palpable tumour justified immediate operation.

At operation on July 21, 1954, the abdomen was opened through a right lower rectus-splitting incision. Free fluid was found in the peritoneal cavity, and an extraordinary looking tumour presented, involving about 30 cm. of the mesentery of the ileum some 60 cm. from the ileocæcal valve (*Fig.* 120).

Multiple cysts of varying size and colour were embedded in a thickened segment of the mesentery, approaching but not involving the antimesenteric border of the ileum. Some neighbouring mesenteric nodes were enlarged and swollen; petechial hæmorrhages were scattered on the surface of the lesion, and many of the cysts contained dark sanguineous fluid.

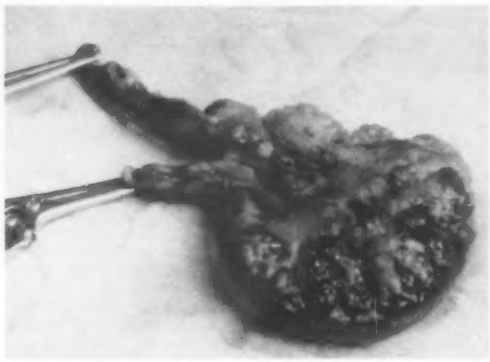

FIG. 120.—Tumour delivered at operation.

About 60 cm. of the bowel and a wedge segment of the mesentery were excised; a side-to-side anastomosis restored the bowel continuity. Convalescence was uneventful and the patient was discharged well on the fourteenth post-operative day. He has remained fit and well for 4 years following operation.

The pathological report by Dr. J. Stewart on the section of the tumour was: "The specimen is 60 cm. in length of small bowel with an extensive angiomatous malformation in the mesentery close to the bowel, which is, however, intrinsically normal. Most of the angiomatous vessels contain blood. Histologically (*Fig.* 121) this is a lymphangioma, into which there has been hæmorrhage, presumably associated with volvulus or some other disturbance of the circulation. The bowel is not entirely unaffected, dilated lymph-channels being present in the muscularis. That the lesion is a developmental anomaly and not acquired elephantiasis is confirmed by the observation that the mucosal lymphatics are unaffected. There is no evidence of malignancy."

DISCUSSION

Study of European and American literature shows a paucity of such cases. Only a few have been described. Hermann and Soloff (1935) described 2 cases in children; MacNab and Menzies (1949) described one case treated by marsupialization of the tumour; while Woolridge (1954) described another. Waters (1946) points out that mesenteric lymphatic lesions are the rarest in the abdomen, a view which Rayford (1932) supported since he found only one case in 11,500 autopsies.

Diagnosis of this type of tumour before operation seems to be impossible. However, a mobile cystic mass

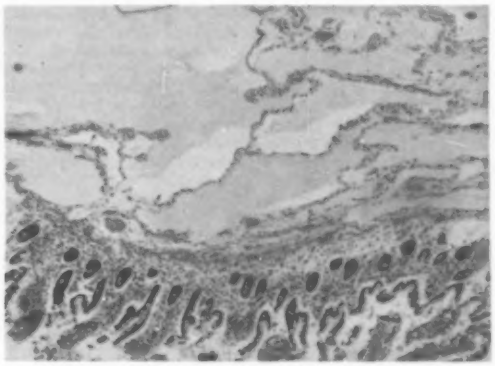

FIG. 121.—Intact epithelium of the small intestine is seen above, lymphangiomatous spaces containing pale-staining eosinophilic fluid occupying the lower part of the field. Hæmalum and eosin. (× 47·5.)

associated with obstructive symptoms, particularly in the male, where ovarian cyst can be excluded, is suggestive of such a lymphatic lesion.

From the practical point of view the aetiology is overshadowed by the simple nature of the lesion and the ease of cure. Marsupialization seems unnecessary, as the method of primary excision and anastomosis is simple and curative.

SUMMARY

A case of lymphangioma of the mesentery is described. This was treated by excision and side-to-side anastomosis. Convalescence was uneventful, the patient being discharged on the fourteenth post-operative day. He is alive and well four years after operation.

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REVIEWS AND NOTICES OF BOOKS

Les Cavités Cardiaques. By E. HENRY, R. COURBIER, and P. ROCHU. 9½ × 6½ in. Pp. 176 + xii, with 196 illustrations. 1959. Paris: Masson et Cie. 3200 fr.

THIS book on the surgical anatomy of the heart comes from the Faculty of Medicine at Marseilles. The authors have based their work partly on studies made in the Department of Anatomy and partly on observations made during cardiac operations performed in the surgical service of Professor R. de Vernejoul. They give detailed descriptions of the cardiac chambers, septa, valves, and outflow tracts. They outline the extent of examination made possible by the commonly used surgical exposures, by digital exploration of the cavities, and by the techniques of open-heart surgery.

The book is well produced, but the illustrations are mostly diagrams and drawings. There are some photographs and a few radiographs of specimens whose salient features have been indicated by radio-opaque markers. The diagrams and drawings are good, but some of the photographs are not clear. Fifty pages of text and ninety-five diagrams are devoted to embryology and the chief types of congenital defects. The description of the coronary vessels is admittedly brief. In a book devoted to open-heart surgery this section could with advantage have been amplified. There is no bibliography, and the reference to "Spitberd" in the embryological section is almost certainly a misprint for "Spitzer".

Some of the authors' remarks call for comment. It is stated that only bilateral thoracotomy with transverse division of the sternum is suitable for open-heart surgery, and scant attention is given to median sternotomy. The latter has many advantages and is now being used extensively by surgeons in many parts of the world. Likewise, though division of the septal cusp of the tricuspid valve is used by some surgeons in the trans-atrial approach to the ventricular septum, few will agree that separation of this cusp from its papillary muscles is necessary for repair of a ventricular septal defect.

Despite these criticisms, the authors have produced an attractive and readable book which is worthy of its alternative title, "An Anatomical Introduction to Intracardiac Surgery".

Die Behandlung von Hiatusheinien und Refluxösophagitis mit Gastropexie und Fundoplicatio. By R. NISSEN and M. ROSSETTI. 9½ × 6½ in. Pp. 153, with 56 illustrations. 1959. Stuttgart: Georg Thieme. D.M.49.50.

As is well known, hiatus hernia associated with reflux-œsophagitis constitutes a prime example of surgical reconstruction failing to guarantee restoration of function and cure of symptoms.

The attractiveness of the authors' approach to this problem would appear to rest on their claim that a much simplified trans-abdominal operation (not applicable to short œsophagus) succeeds in arresting the reflux.

The cogency of their contribution is weakened theoretically in several ways: more than 20 per cent of patients in the series had no reflux-œsophagitis, but have not been excluded from consideration. The authors will agree that operative treatment of para-œsophageal hiatus hernia poses much less difficulty. Further, follow-up of less than one year in 40 per cent of patients with reflux may arouse scepticism. Finally, upward of 10 per cent of patients with reflux were coincidentally subjected to vagotomy or subtotal gastrectomy; the addition of vagotomy to the operation may not detract from the simplicity of the over-all procedure, but why should it not take pride of place if the symptoms are then cured?

Interested surgeons might like to give this method, clearly set out and well illustrated, a trial. It is to be hoped that this monograph will secure the entrée for Prof. Nissen to the British literature, which travels the Atlantic with ease but seems to be daunted altogether by the channel-crossing.

A Synopsis of Anæsthesia. By J. ALFRED LEE, M.R.C.S., L.R.C.P., M.M.S.A., F.F.A.R.C.S., D.A., Senior Consultant Anæsthetist to the Southend-on-Sea Hospital. Fourth edition. 7½ × 4½ in. Pp. 616 + vi, with 72 illustrations. 1959. Bristol: John Wright and Sons Ltd. 27s. 6d.

THE new fourth edition of this very practical synopsis has been very thoroughly revised and considerable new matter has been added.

The field of anæsthesia is so vast to-day and changes take place so rapidly in the use of new drugs and new techniques. Anæsthesia plays such an important part in every branch of surgery in these modern times. Now that there is a higher diploma in anæsthesia this synopsis should prove invaluable to those doctors who are preparing for this examination, for the book is in itself a very practical scaffolding on which to answer any question dealing with anæsthesia.

A most valuable book for quick revision and, as such, it will be of value to both undergraduates and post-graduates alike.

Year Book of Neurology, Psychiatry and Neurosurgery. Edited by ROLAND P. MACKAY, M.D., S. BERNARD WORTIS, M.D., and OSCAR SUGAR, M.D. 7½ × 5 in. Pp. 623, with 109 illustrations. 1959. Chicago: The Year Book Publishers Inc. (London: Interscience Publishers Ltd.) 64s.

THIS is one of the fifteen year books comprising the Practical Medicine Series which comes from this publishing house.

R. P. Mackay, the editor of the section on neurology, tells us that the most significant developments of the year in his subject are in the field of neurotropic viruses, of which the commoner poliomyelitic varieties are only the most neurotropic, while others may cause meningo-radiculoneuritis, epidemic myalgia, and other syndromes. The safety and effectiveness of the Salk vaccine now appear to be well established and the immunity conferred by it as enduring as that from natural infection.

S. Bernard Wortis, who edits the section on psychiatry, finds that as the newer psychotropic drugs are being developed, prefrontal lobotomy for psychiatric patients is being performed less often. In Russia the procedure has been forbidden since 1950.

Oscar Sugar edits the section on neurosurgery and sums up the year's advances as continued interest in such questions as hibernation, the reduction of brain swelling, hypophysectomy for metastatic cancer, and vascular grafting for arterial occlusive disease. Chemopallidectomy has less space than might perhaps have been expected for this still new procedure. There is a brief reference to the carpal tunnel syndrome which was not mentioned in last year's volume, but the condition does not appear to attract the attention in America that it does in this country and there are no references to cervical spondylosis. In a compact review of the year's many contributions, however, these are perhaps minor omissions and the editors are to be congratulated on their condensation and the pithy comments which they have made on certain of them.

These year books are not only invaluable to busy practitioners of the specialties to which they relate but they also have much of interest to practitioners generally; this volume on the nervous system especially so.

Wound Healing and Tissue Repair. Edited by W. BRADFORD PATTERSON. 9 × 5¾ in. Pp. 83 + xii, with 25 illustrations. 1959. Chicago: University of Chicago Press. (London: Cambridge University Press.) 21s.

THIS volume is one of ten reports of a series of conferences on developmental biology held in various educational centres in North America under the ægis of the National Academy of Sciences and the National Research Council of the United States. The chairman of the whole series was Paul Weiss, head of the Department of Developmental Biology of the Rockefeller Institute, New York. The conferences were attended by nearly 300 American and 54 visiting scientists from 19 other countries.

Wound healing, the last subject to be dealt with in the series, was discussed by a panel of 30 research workers representing every relevant scientific discipline. Francis D. Moore and W. Bradford Patterson, both of the Harvard Medical School, were the Chairman and Reporter respectively.

The subjects discussed were—the biological aspects of wound healing, the cellular components of the wound, the ground substance, the relation between healing and sulphur metabolism, the systemic factors involved, the healing of bone, and abnormal healing. The discussions attained a high scientific standard, and after reading them it becomes clear that to appreciate their quality the general reader would be well advised to read the reports of at least four of the preceding discussions in the series—those dealing with the biology of regeneration in vertebrates, cyto-differentiation, tissue proliferation, and mitogenesis.

With this background the report will certainly provide the surgeon and laboratory worker with an up-to-date account of the progress made and the very substantial obstacles encountered in the application of modern scientific conceptions and methods to the many unsolved problems presented by the healing wound.

The discussions have been exceptionally well edited and well purged of irrelevant detail.

Quadri Arteriografici. By E. MALAN and collaborators. 11½ × 8 in. Pp. 124, fully illustrated. 1959. Torino: Edizioni Minerva Medica. L.5000.

THIS atlas is derived from the authors' series of over three thousand arteriograms carried out in the past ten years. After a brief outline of the history and technical details of the subject, there follows a clear account of the arteriographic anatomy of the abdomen and both limbs. The remainder of the book comprises seven sections illustrating the appearances seen in juvenile and senile obliterative disease, arterial aneurysms, congenital and post-traumatic arteriovenous fistulæ, Raynaud's syndrome, and finally a syndrome newly described by Malan, to which he attaches his name (Malan's syndrome). This consists of dilatation of the arteriovenous connexions in the sole of the foot in association with chronic venous stasis (varicose or post-phlebitic). The dilatation causes increased flow in the arterial trunk supplying the affected area, and this takes place at the expense of neighbouring vessels. If the dilated communications are in the posterior part of the sole (supplied by the posterior tibial artery), there is consequent ischæmia in the anterior tibial territory. If the dilatations are in the forefoot, the reverse applies, and if throughout the sole, both tibials have an augmented flow at the expense of the peroneal. All these possibilities and the dilated communications themselves are beautifully illustrated in a series of superb arteriograms (slightly confused, however, by the reversal of the titles between pp. 108 and 110).

Similar conclusions were reported in this country by Brewer in 1950. Malan's arteriograms confirm his work and seem to establish the phenomenon beyond further question.

Throughout the book the arteriograms are clear and well chosen, and form a useful compilation illustrative of all the common manifestations of arterial disease in the lower abdomen and limbs. The quality of the half-tone reproductions is excellent, though unfortunately the originals are positive prints and some appear to have been rather heavily re-touched. Accompanying details of the injection methods and exposure times used greatly increase the value of the illustrations. Altogether the book is a helpful guide to peripheral arteriographic appearances and a creditable record of personal achievement.

Surgery of the Foot. By HENRI L. DU VRIES, M.D. Clinical Instructor in Surgery, Chicago Medical School. 10 × 7 in. Pp. 494, with 403 figures. 1959. St. Louis: C. V. Mosby Co. (London: Henry Kimpton.) 93s. 6d.

THE surgery of the foot covers a wide field and often a neglected field at that. Although there are other books devoted to the surgery of foot conditions there is yet always room for others if they are comprehensive and practical.

Dr. Du Vries has written a useful volume which is well illustrated and is exhaustive. Many of the conditions are rare, but this makes the book a reliable reference volume.

To-day minor conditions of the foot are neglected or not treated at all, while some conditions only come under the surgeon's care when they are far advanced.

This is a book crammed with information about common conditions.

Synopsis of Treatment of Anorectal Diseases. By STUART T. ROSS, M.D., F.A.C.S., F.I.C.S. 7¼ × 5 in. Pp. 240, with 79 illustrations. 1959. St. Louis: C. V. Mosby Co. (London: Henry Kimpton.) 48s. 6d.

THIS small monograph dealing with the treatment of diseases of the rectum and anus gives an admirable account of these all too common diseases. It should prove of real value to the student and practitioner.

Introduction to Surgery. Fourth edition. By VIRGINIA K. FRANTZ, M.D., and HAROLD D. HARVEY, M.D. 7¼ × 5 in. Pp. 335 + xiv, with 13 illustrations. 1959. New York: Oxford University Press. 48s.

THE fourth edition of this handy volume has been very thoroughly revised and brought up to date. Several new contributors have been added, but the size and format of the book remains unchanged. An excellent volume for the student who is just commencing his clinical work in the wards.

Diseases of the Nose, Throat and Ear. Edited by CHEVALIER JACKSON, M.D., Sc.D., LL.D., L.H.D., F.A.C.S., and CHEVALIER L. JACKSON, M.D., M.Sc., F.A.C.S. Second edition. 10 × 6¾ in. Pp. 886 + xviii, with 110 illustrations. 1959. Philadelphia and London: W. B. Saunders Co. 140s.

THE second edition of this well-known work will be welcomed by the medical profession, for it sets out current surgical opinion in America concerning the diseases of the ear, nose, and throat. The Jacksons themselves have been helped by the collaboration of

sixty-one well-known American surgeons who have made these branches of surgery their special field.

The book is profusely illustrated with excellent drawings, radiographs, and photomicrographs.

As one would expect with two such expert endoscopists, that section of the book which deals with peroral endoscopy is superbly written and illustrated. The name Chevalier Jackson carries the same respect in England as it does in America in things pertaining to diseases of the ear, nose, and throat.

Like the previous edition, this is an outstanding work and every medical library should have a copy for ready reference.

Trauma. By HARRISON L. MCLAUGHLIN, M.D., Professor of Clinical Orthopædic Surgery, College of Physicians and Surgeons, Columbia University. 10 × 6½ in. Pp. 784 + xiv, fully illustrated. 1959. Philadelphia and London: W. B. Saunders Co. 126s.

WE are living in an age when trauma makes more and more inroads on surgical practice. It may be said that trauma cuts across all branches of medicine and therefore the surgery of trauma becomes more important each successive year. This large volume largely written by orthopædic surgeons covers the immense field of trauma in a very practical fashion. The book is well illustrated and the problem of treatment is well presented.

As one would expect, the subject of fractures is dealt with in an excellent way.

Students, resident medical officers, and practitioners will find this book both practical and useful.

Peripheral Vascular Diseases: An Objective Approach. By TRAVIS WINSOR, M.D., F.A.C.P., Assistant Clinical Professor of Medicine, University of Southern California School of Medicine, Los Angeles. 9 × 5¾ in. Pp. 845 + xviii, with 435 illustrations. 1959. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 125s.

PERIPHERAL vascular diseases are all too common to-day and it is desirable that all surgeons should have a sound knowledge of this important subject.

Professor Winsor has made a modern objective approach to this problem in his recent book. His clinical acumen and the use of numerous techniques employing electronic instruments has resulted in a very readable and interesting book. The illustrations throughout are excellent; the majority are original and are of real assistance to the reader.

The operation of sympathectomy is given its proper place and the various operative techniques are well described.

There can be no doubt that this book covers the whole field of peripheral vascular diseases with a vigorous freshness created by the matured clinical background of the author.

This is a book which will be popular to both physician and surgeon alike.

The Life and Times of Sir Charles Hastings. By WILLIAM H. MCMENEMEY, M.A., D.M., F.R.C.P., D.P.M. 8¾ × 6½ in. Pp. 516 + xii, with 30 plates. 1959. Edinburgh and London: E. & S. Livingstone Ltd. 50s.

THIS is an excellent volume depicting the life and times of the founder of the British Medical Association. Dr. McMenemey has performed his task well and has given the profession a first-class description of the medical affairs in Worcester in the early part of last century. It is true that the author spent several years as pathologist to the Worcester Royal Infirmary and he carried out his researches into the activities of Sir Charles Hastings with great zeal. In 1951 Dr. McMenemey delivered the first Sir Charles Hastings Memorial Oration and this volume is an elaboration of that lecture.

The style of the author is lucid and interesting, and he has the happy knack of making his reader live in the past while reading the book.

Dr. McMenemey has been very industrious in obtaining photographs and prints of prominent medical men who came into contact with Sir Charles Hastings during his lifetime. One cannot help feeling that the compilation of this excellent biography has been a labour of love because the author's heart was in the work.

This volume will enhance the already numerous historical biographies which have been published by Messrs. E. & S. Livingstone.

We consider the price of the volume to be very moderate.

The Year Book of Orthopædics and Traumatic Surgery. Edited by EDWARD L. COMPERE, M.D., F.A.C.S., F.I.C.S., with a section on Plastic Surgery edited by NEAL OWENS, M.D., F.A.C.S., F.I.C.S. 7½ × 5 in. Pp. 445, with 227 illustrations. 1959. Chicago: The Year Book Publishers Inc. (London: Interscience Publishers Ltd.) 57s.

THIS useful volume is packed full of information gleaned from the many and various articles on orthopædic subjects which have appeared in the international medical press during the last year.

It appears that orthopædic surgeons to-day are inclined to resort to surgery for many conditions where ten years ago conservative treatment would have been adopted. One example will suffice—namely, nailing the femoral head when dealing with a slipped epiphysis of the hip-joint.

Neal Owens has edited a section on plastic surgery which is excellent. Orthopædic and plastic surgeons overlap each other in the treatment of burns, contractures, and congenital deformities.

Pocket Book of Proprietary Drugs. Compiled by ALISTAIR G. CRUIKSHANK, F.R.C.P.E., and CORNELIUS STEWART, L.R.C.P. & S.E., M.P.S. 6 × 4¼ in. Pp. 236. 1959. Edinburgh and London: E. & S. Livingstone Ltd. 10s. 6d.

THIS is a most useful pocket book and will prove invaluable to every branch of the medical profession, for it contains much which is difficult to obtain elsewhere.

To have in one small volume the names, manufacturers, compositions, indications, packs and dosages of some fifteen hundred preparations offered by the pharmaceutical industry for prescription by the medical profession is of real value and usefulness.

This is a book that clinicians, students, and practitioners will carry about with them and make constant reference to its pages.

A most useful production. An interleaved edition is also available.

Plaies de la Main. By R. SOUQUET and A. R. CHANCHOLLE, The Faculty of Medicine, Toulouse. 9½ × 6 in. Pp. 293, with 131 illustrations. 1959. Paris: G. Doin & Cie. 3600 fr.

THIS is an excellent monograph on the diagnosis and management of hand injuries based on the experience of the authors in the University Hospitals of Toulouse. The first chapter deals with the causes of industrial accidents

and discusses the very serious social problems involved. Subsequent chapters deal with the problems of skin loss and injuries to the tendons, nerves, and bony skeleton. There are good sections on the treatment of burns and amputations and a very useful short chapter on the role of physiotherapy and rehabilitation by Dr. Despeyroux.

This outline of the management of hand injuries follows very closely accepted British practice. The book is beautifully produced and the illustrations both in black and white and in colour are a credit to the authors and their publishers. There will undoubtedly be several reprints of this book and perhaps the publishers would consider a more robust binding for future editions. The value of the book would also be enhanced if more care were taken with the bibliography: full names and initials should be given and several misprints corrected. It would also be an advantage when authors' names are mentioned in the text to give the year of publication or some other method of tracing the paper to which reference can be made in the bibliography at the end of the book.

Surgery of the Ear. By GEORGE E. SHAMBAUGH, jun., M.D., Professor of Otolaryngology, Northwestern University Medical School. 10¾ × 7¾ in. Pp. 669 + viii, with 326 illustrations. 1959. Philadelphia and London: W. B. Saunders Co. 152s. 6d.

THIS is probably one of the largest and most comprehensive volumes ever written in the English language on the surgery of the ear. It is quite true to say that no branch of surgery has been altered more profoundly, advanced so rapidly, and benefited so greatly by the use of penicillin and the antibiotics.

The operative procedures of twenty years ago have been completely altered and some operations performed then are no longer carried out.

This volume deals with the whole field of the surgery of the ear.

We are pleased to see an excellent picture of Mr. Terence Cawthorne in that part of the book which deals with infections of the ear. We would all agree with his statement: "It is essential to regard chemo- and biotherapy on the one hand and surgery on the other not in any way as rivals but as being complementary to one another." This practical statement applies to every branch of surgery.

That part of the book which deals with the various techniques of the fenestration operation is particularly clear and interesting.

The illustrations throughout the book are of a high order and do help the reader to understand the text more easily.

A magnificent production which will take its place as the best book on the surgery of the ear.

A Synopsis of Skin Diseases. By BETHEL SOLOMONS, jun., M.A., M.D., F.R.C.P.I., Consultant Dermatologist, Chelmsford and Essex General Hospital. 7¼ × 4¾ in. Pp. 293, illustrated. 1959. Bristol: John Wright & Sons Ltd. 30s.

THERE can be little doubt that dermatology is a very difficult branch of medicine, and, it is often, as far as the student is concerned, a neglected branch. However, it is a most important branch, especially for the busy practitioner.

Dr. Solomons has written a most useful book, for he has produced in synopsis form a very serviceable and interesting volume. Every branch of dermatology has been covered. There are some excellent black and white drawings illustrating the regional sites of common skin diseases. The comprehensive list of occupational causes of contact dermatitis is most useful for ready reference.

This is the book the general practitioner has been looking for, and the student will find it invaluable for quick revision of the whole subject of dermatology prior to his final examinations.

Surgery for Nurses. By JAMES MORONEY, M.B., Ch.B., F.R.C.S.(Eng.), L.R.C.P.(Lond.), Visiting Consultant Surgeon, Clatterbridge General Hospital, Cheshire, and Broadgreen Hospital, Liverpool. Sixth edition. 8¼ × 5¼ in. Pp. 751 + xii, with 618 illustrations. 1959. Edinburgh and London: E. & S. Livingstone Ltd. 30s.

THE fact that this new sixth edition appears a year after the fifth edition speaks for itself. This book contains all the surgery that it is necessary for the modern nurse to know in order to pass the State examination.

This new edition is quite up to date and contains many new illustrations. A very practical and popular book.

An Introduction to Surgery for Dental Students. By R. P. JEPSON, F.R.C.S., Professor of Surgery, University of Adelaide, and B. N. CATCHPOLE, F.R.C.S., Lecturer in Surgery, University of Sheffield. 8¼ × 5¼ in. Pp. 166 + x, with 53 illustrations. 1959. London: The English Universities Press Ltd. 21s.

THIS is a useful book for the dental student for it gives him an excellent introduction to the principles of general surgery. It is these principles which are so often neglected in dental schools, the students being coached in dental surgery and the principles of surgery being ignored.

As one would expect, the larger part of the book is concerned with a description of the diseases affecting the various regions of the head and neck, of which the dental surgeon is expected to have some knowledge.

A useful little book.

Notable Names in Medicine and Surgery. By HAMILTON BAILEY, F.R.C.S.(Eng.), F.A.C.S., F.R.S.(Edin.), and W. J. BISHOP, F.L.A. Third edition. 8¼ × 5¼ in. Pp. 216 + xiv, with 79 portraits and 162 other illustrations. 1959. London: H. K. Lewis & Co. Ltd. 35s.

THE third edition of this little book gives an excellent account, with numerous pictures, of eminent physicians, surgeons, and scientists of the past.

It should prove of real value to the student and practitioner who require a reference volume to historical medical names.

To-day there is a tendency, which we sincerely regret, to leave out names of anatomists and scientists which are associated with structures or diseases. This book will help to correct this modern tendency in medical education.

Physiology of Cardiac Surgery. By FRANK GOLLAN, M.D., Assistant Director of Professional Services for Research, Veterans Administration Hospital, Nashville, Tennessee. 8¼ × 5¼ in. Pp. 96 + x, with 18 illustrations. 1959. Springfield, Illinois: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 34s.

THIS is a very practical book dealing with the important subjects of hypothermia, extracorporeal circulation, and extracorporeal cooling. These are subjects of immense interest to the cardiac surgeon of to-day and allow operations on septal defects of the heart to be operated upon in a dry field.

The author, who has published a spate of articles in America on these subjects, discusses in this book the differences between spontaneous hibernation and induced hypothermia in anæsthetized animals and man. Gollan gives clear reasons for the limitations of induced hypothermia by peripheral cooling.

This book places extracorporeal cooling in its proper perspective in the medical field to-day.

An excellent bibliography at the end of the book will be found of value by all those who are interested in this branch of research.

Autolyse-Krankheiten in der Chirurgie. By L. KOSLOWSKI. 9¼ × 6¼ in. Pp. 159 + viii, with 60 illustrations. 1959. Stuttgart: Georg Thieme. D.M.19.80.

MODERN advances in medicine, when measured in terms of improved life-salvage, are truly spectacular. At the same time, fatalities occurring in the face of medical progress have assumed added importance, and are stimulating increased thought.

In a clinical and experimental study, the author postulates that failure to overcome disease or injury, despite the best that medical treatment can now offer, is often an effect of spontaneous tissue-disintegration on the metabolic processes. He suggests that such tissue-disintegration, or autolysis, evokes a distinctive disease-entity—"autolysis-disease"—whatever the underlying cause.

This closely-reasoned and well-documented monograph, though written in a style not easy to assimilate, makes a valuable contribution towards closing a baffling gap in medical, but especially traumatological, knowledge. From a practical point of view, the ideas advanced to combat "autolysis-disease" are interesting and provocative.

Malignant Disease, University College Hospital, 1946-1950. Compiled by the Cancer Follow-up Department, University College Hospital, London. Pp. 631 + vii. Wilding and Son Ltd., Shrewsbury, England, 1959. 100s.

THIS is an excellent catalogue of all cases of malignant disease, reticulosis, leukæmia, mixed salivary tumour, adenoma of the bronchus, and papilloma of the bladder, seen in the wards and out-patient departments of University College Hospital, London, between Jan. 1, 1946, and Dec. 31, 1950.

This large volume contains a wealth of material which should prove of real value to all who are interested in the successful treatment of malignant disease.

The whole lay-out of the book is most practical and it should serve as a useful guide for the future 'follow-up' of malignant cases.

Atlas de Techniques Chirurgicales. By R. MICHEL-BÉCHET. 14¾ × 10¾ in. Pp. 580, with 1125 illustrations. 1958. Paris: C. Doin et Cie. 25,000 fr.

AT every intellectual level, and not least in the field of medical education, there is a steady trend towards greater use of pictorial methods of communication. While atlases of anatomy, retinoscopy, and so forth are already familiar, at least two new developments, the atlas of operative surgery and the closed-circuit television demonstration, may be claimed for the present decade. It is of course true that Cutler and Zollinger's *Atlas of Surgical Operations* was first published in 1939, followed, ten years later, by a second edition, but with this exception, works in the same genre have appeared in English mainly since 1954.

Michel-Béchet's atlas, avowedly inspired by Cutler and Zollinger's, is evidently the first of its kind in French and the author expresses the hope that it will be of assistance to French surgeons by enabling them to gain a rapid yet exact conception of the details of the operations which they are about to perform. A further and very great advantage of pictorial presentation not mentioned by the author is its independence of language barriers. By the perusal of this book, not only French surgeons but British ones as well may inform themselves of the current methods of French surgical practice with the minimum of effort and confusion. At a time when surgeons in the English-speaking world are so scantily informed about the work of the various Continental schools, there seems to be a real need for more such atlases from all parts of Europe.

The work is divided into four main sections dealing with the neck, thorax, abdomen, and pelvis (including gynæcology). There are no sections on facial, neurological, orthopædic, plastic, urological, or vascular surgery, and the section on the thorax deals only with general surgeons' operations, omitting the surgery of the lungs, heart, and great vessels. Within these limits, the choice of material is wide, though not exhaustive. Many of the methods illustrated will be familiar to British surgeons even if the names attached to them are not, but there is also much that will be strange.

The first section (neck) includes descriptions of parathyroidectomy, subtotal thyroidectomy, and thyroidectomy for carcinoma; block dissection of the neck by Dargent's method from above downwards, preserving the accessory nerve; operations on the cervical œsophagus, trachea, phrenic nerve, carotid body, cervical rib, and stellate ganglion. Parotidectomy and orbital decompression are also included.

The section on the thorax is the shortest of the four and includes radical mastectomy (breast before axilla), thoracotomy, and operations on the vagus, lower œsophagus, and diaphragmatic hiatus, together with a method of prethoracic œsophagoplasty using the right half of the colon. It will interest non-French-speaking readers to learn that transthoracic vagotomy is known as 'Dragstedt par voie thoracique'.

The third section on the abdomen is the longest and comprises approximately half the book. It describes all the familiar major visceral operations with many ingenious details unknown here. Great prominence is given to re-peritonization of denuded surfaces and exclusion of suture lines from the peritoneal cavity. The inclusion of intestinal anastomosis by button and six methods of gastrostomy seem rather old-fashioned, as does the opinion that, after resecting gut from a strangulated hernia, it is necessary to drain the dilated segment by an enterostomy to prevent grave and often fatal intoxication from subsequent re-absorption of its fluid contents. For inguinal hernia, the methods illustrated are those known here by the names of Bassini, Halsted, Bloodgood, and McVey (but differently called in France) together with cutis grafting. Biliary tract surgery is well covered and in this field the outlook is much less in-bred than elsewhere; so much so in fact, that notable French work escapes mention. By contrast, the approach to carcinoma of the rectum is essentially French and shows great preoccupation with the conservation of anal function. d'Allaines' work is prominent and abdominosacral and sacral methods are also given. Classical abdominoperineal resection is shown, together with methods of combining it with vaginal hysterectomy and with perineal colostomy. Perineo-abdominal and synchronous combined methods are not included.

The illustrations by P. Contier are well drawn and for the most part easy to follow. The text is reduced to a bare minimum (much less than in comparable works in English) and many of the figures bear no labelling at all, which may partly explain why Fig. 9 on p. 45 is reproduced upside down. The subject is shown purely as a matter of cutting and stitching with little guidance on pre- and post-operative care or on those matters which fall under

the general heading of surgical judgement. There is no bibliography or index, though the table of contents, printed at the back of the book, is very detailed. Foreign authors sometimes appear lightly disguised, as for example Blackemon and Whippe (authorities on porto-caval anastomosis), Babcok and Bacon (excision of the rectum), and Senn Kaderre (thus in the table of contents, Sen Kaderre in the text—gastrostomy).

The book is luxuriously bound and jacketed and printed on art paper throughout. It is expensive, even by French standards.

The Mast Cells. By JAMES F. RILEY, M.B., Ch.B., M.D., Ph.D., D.M.R.T., F.R.C.S.E., Consultant Radiotherapist, Royal Infirmary, Dundee. 9¾ × 6½ in. Pp. 182 + x, with 65 illustrations. 1959. Edinburgh and London: E. & S. Livingstone Ltd. 30s.

EIGHTY years ago Ehrlich first described the mast cells or 'basophils' of the blood and tissues, and he made special reference to the large, brilliantly-staining basophil granules tightly packing their cytoplasm. They were shown by the Scandinavians, twenty-two years ago, to contain heparin—a discovery which suggested that they may be the source of a physiological anticoagulant.

While this generalization has not been substantiated it was then demonstrated by the author of this book that tissue basophils are rich in histamine, as are those of the circulating blood, and that histamine liberators cause swelling and degranulation of tissue mast cells. Dr. Riley has also shown that basophils are present in large numbers in serous membranes, in perivascular connective tissue, and in healing tissues; whilst in the mouse and rat they appear to be a rich source of 5-hydroxytryptamine.

The book tells the story of the author's absorbing interest in these fascinating cells over the last thirty years. During this time he has made substantial contributions to our knowledge of the function of mast cells which show every indication as being of fundamental importance. His book is attractively written, superbly illustrated, and remarkably easy to read. His enthusiasm for this cell is infectious.

British Medical Bulletin. Volume 15, Number 2, May 1959. Blood Groups. 11 × 8½ in. Pp. 174 + xii. Published by the Medical Department, The British Council. 20s.

THIS number of the *British Medical Bulletin* is a symposium of fourteen papers dealing exclusively with blood groups. It was planned by a Committee whose Chairman was Dr. A. E. Mourant, and Dr. K. L. G. Goldsmith acted as the scientific secretary.

The papers cover a remarkably wide field, including biochemistry, immunology, genetics, pathology, pædiatrics, and obstetrics, as well as basic biology and anthropology. A good deal of the research reported has been carried out by British workers, who describe their own problems. Other papers are reviews of modern knowledge.

Without exception all the articles are authoritative, well balanced, of absorbing interest, and well documented.

Smoking and Lung Cancer. By T. W. LEES, M.D., Law Hospital, Carluke, Lanarkshire. 8½ × 5½ in. Pp. 32. 1959. Published by the Author.

THIS slim, paper-backed pamphlet makes interesting reading. It is written and published by a pathologist who has never smoked a cigarette or a pipe. From a statistical survey which is described in simple and understandable language, he infers that "there is no good evidence that smoking causes lung cancer".

It would appear that the arguments used in this small monograph should be examined by the statisticians who have reached the opposite conclusion and that their deductions should be made public.

Le Risque Opératoire en Chirurgie Bilio-pancréatique. By YVES SALEMBIER. 9¾ × 6½ in. Pp. 260 + viii, with 2 illustrations. 1959. Paris: Masson et Cie. 2500 fr.

THE author's purpose in writing this book is to analyse and consider the major complications arising within one month of operation on the biliary and pancreatic tracts in the hope of being able more effectively to avoid these, in the future. The overall mortality of the author's thousand cases was 7·5 per cent. This mortality-rate is broken down by cause of death, but is not analysed according to preoperative diagnosis or nature of operation. Thus bilio-pancreatic surgery is treated throughout as though it were a homogeneous entity. There are short case-histories of 46 of the 75 fatal cases.

The book is divided into four parts. The first, which is largely physiological, deals with such subjects as hepatic, pancreatic, and renal function and the appropriate methods of testing these; blood volume, anæmia, and hypoproteinæmia. The effects of age, sex, obesity, and the nature of the lesion are also considered and a points system of assessing operative risk is proposed.

The second part outlines patient care before, during, and after operation. Besides the correction of anæmia, hypoproteinæmia, vitamin depletion, and defective blood-volume, the author recommends that all cases, even straightforward cholecystectomies, should be prepared for at least two days with intravenous injections of hypertonic glucose, serum, insulin, acetyl methionine, and vitamins B_1, B_2, and B_6, as well as intramuscular ascorbic acid, liver extract, and vitamin K_1. In addition, all patients over 65 receive testosterone propionate for 6 to 8 days before operation, together with pituitary growth hormone, glutamic acid, and placental extracts. Post-operative care includes the control of fluid and electrolyte balance, and the relevant basic information is set forth at great length.

In the third and fourth sections the author records his personal experience, reviewing with short case-histories the complications encountered in his 75 fatal cases. Notable among these is hæmorrhage, which, in spite of polypharmacy, accounted for 12 deaths, a figure not exceeded by any other cause, and equalled or approached only by hepatorenal failure (12 deaths) and malnutrition (10 deaths). Using radio-manometric techniques for exploration of the common bile-duct, re-exploration for residual stones proved necessary in only 5·6 per cent of 170 cases. The methods of detection and extraction mentioned include the cholelithophone, the operating choledochoscope described by Wildegans, and the use of extraction in a matrix of fibrin clot. The author also recommends the use of a sucker passed down the duct.

The book provides an objective record of a large volume of experience in biliary and pancreatic surgery. Although the text makes liberal reference by name to the work of other authorities, there is no bibliography or other indication of the source of these citations. This is not unusual in French monographs.

The Effect of Advancing Age upon the Human Spinal Cord. By L. RAYMOND MORRISON, M.D., with the collaboration of STANLEY COBB and WALTER BAUER. 11 × 8½ in. Pp. 127 + x, fully illustrated. Cambridge, Mass.: Harvard University Press. (London: Oxford University Press.) 45s.

IN mammals there is no satisfactory evidence of regeneration of the divided spinal cord either in fœtuses or adults, but a remarkable degree of recovery of conduction

may occur after extreme compression and deformation of it provided this has been gradual. Long ago Wilfred Trotter was impressed by the great importance of youth in favouring this recovery in the human cord.

As the result of a great deal of painstaking work by the late L. Raymond Morrison, who died in August, 1950, in the course of the preparation of this monograph and by Doctors Stanley Cobb and Walter Bauer, who have now brought it to fruition, we have an informative atlas of the changes in the human spinal cord with ageing. The investigation was begun to ascertain whether in rheumatic diseases the nervous system was, or was not, involved, and as controls were necessary a study was made of the effect of advancing age on the human cord. Observations were made on the cords of 50 patients aged from 10 to 90 who during life had had no neurological symptoms and who came to autopsy in a general hospital. The specimens of the cords were prepared by L. R. Morrison himself, who both described and photographed the changes in nerve-cell, axon, myelin, glia, and connective tissue.

Although little degeneration occurs in the first three decades, even in the second there is some early indication of approaching changes, such as some breaking up of axons. Corpora amylacea appear in the fifth decade and from then on chromatolysis and glial proliferation are progressive and the ependyma of the central canal thickens and may obliterate it. The second part of the atlas shows the features of the different segments of a normal cord of a person aged 24.

This is a valuable atlas with a bibliography but without any index, which perhaps it hardly requires. The authors' expressed hope that by forming a base line, or normal control, for any studies of the human spinal cord in which factors associated with increasing age must be taken into account, it will serve as a valuable source of reference in the study of diseases of the spinal cord, is likely to be fulfilled.

Surgery of the Stomach and Duodenum. By CLAUDE E. WELCH, M.D., D.Sc. (Hon.), Visiting Surgeon, Massachusetts General Hospital. Third edition. 8¼ × 5¼ in. Pp. 405, with 83 plates. 1959. Chicago: Year Book Publishers Inc. (London: Interscience Publishers Ltd.) 74s.

THE new third edition of this well-known handbook of operative surgery has been completely revised and includes significant advances in the surgery of stomach and duodenum that have emerged in the past four years.

Some new illustrations have been added, but the format of the book remains the same. A very useful book for all senior registrars and young surgeons.

A feature of the book is the excellence of the illustrations, the majority of which are line drawings.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.*]

Traumatismes Anciens. Rachis-Membre Inférieur. By R. MERLE and J. O. RAMADIER. 9½ × 6¼ in. Pp. 546 + viii, with 218 illustrations. 1959. Paris: Masson et Cie. 7500 fr.

Coupes Horizontales du Tronc. By RAYMOND ROY-CAMILLE. 16½ × 12½ in. Pp. 122, with 54 radiographs and 54 schémas. 1959. Paris: Masson et Cie. 8000 fr.

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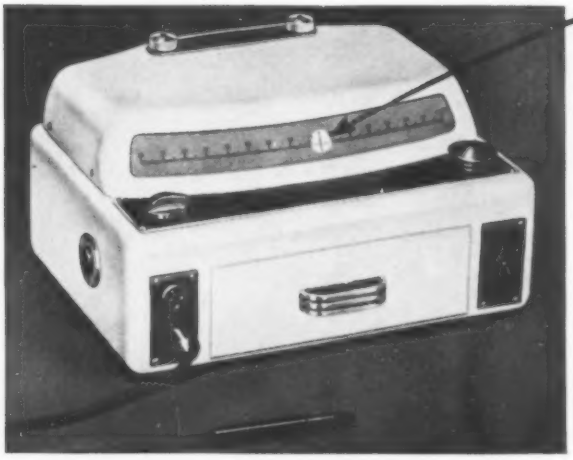

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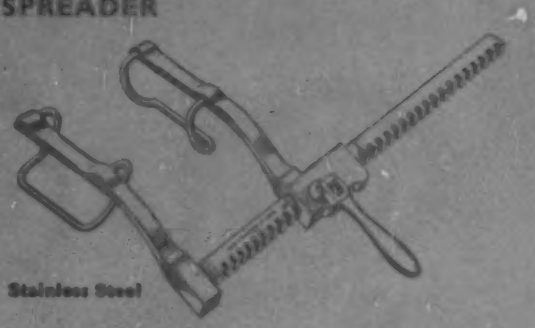

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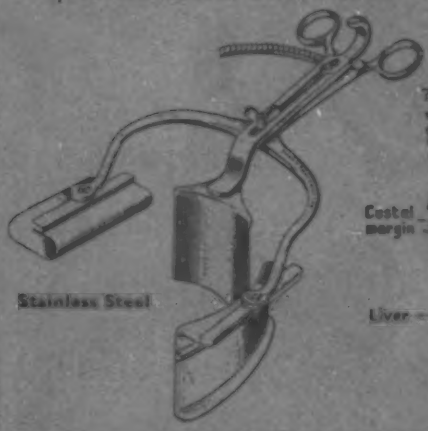

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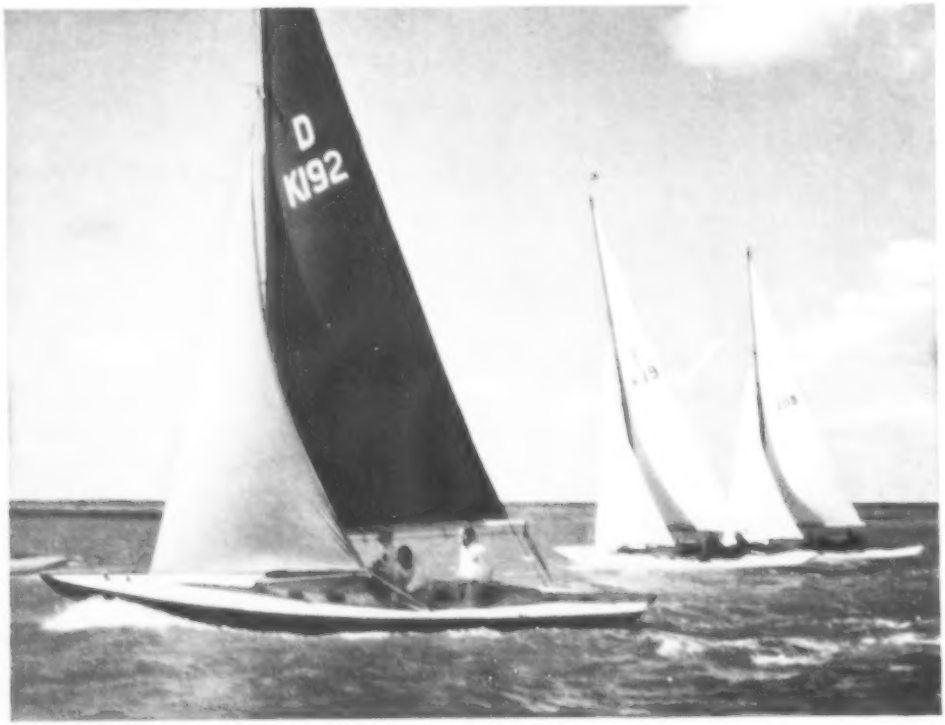

Teamwork Counts

Sailing close to the wind, the crew of this racing Dragon appear relaxed, confident. That's teamwork – however hard pressed, they can still give of their best because they know their craft, they know their winds, and they know and trust each other.

Teamwork counts, too, in radiography. Not simply personnel but films, screens, chemicals, must work together. It's wise, then, to choose materials which are *made* to work together. Kodak materials.

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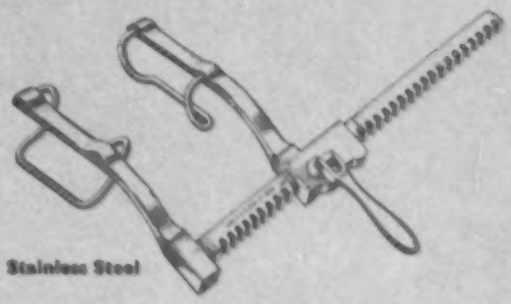

Stainless Steel

**A small size for children is also available.*

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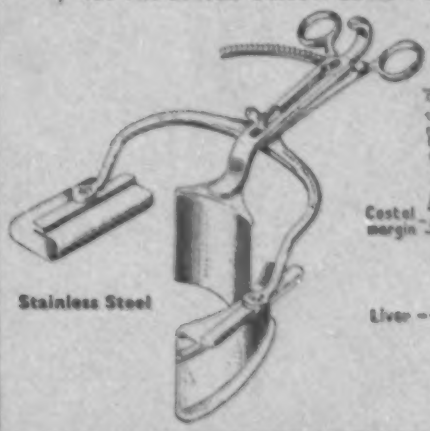
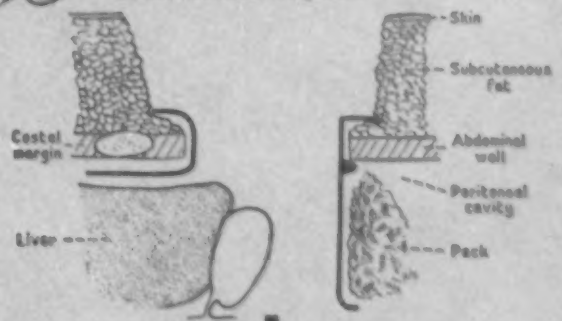
Designed for use with a Kocher incision. Especially useful for very fat patients.

†Vide The Lancet, October 22nd, 1955 p.854.



Stainless Steel

Diagram showing how the two main blades of the retractor are placed



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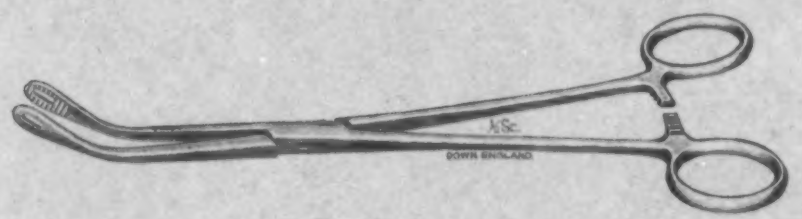
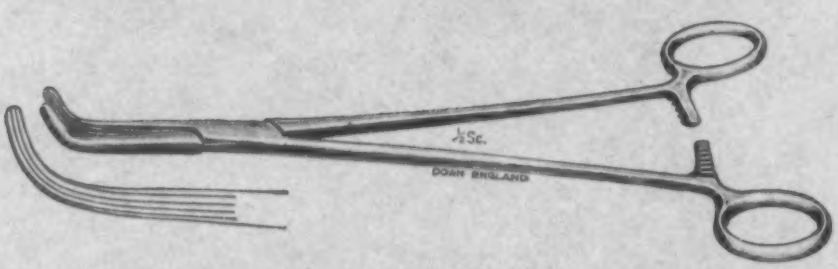
Shiers' modification of Ayre's cervix biopsy scraper



Vant's Uterine Polypus Forceps



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